The relationship between systolic blood pressure on admission and mortality in older patients with heart failure

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Aims

To determine the relationship between admission systolic blood pressure (SBP) and mortality in older patients hospitalized for heart failure (HF) and among various subgroups.

Methods and results

We evaluated the independent association between initial SBP and 30-day and 1-year mortality, and potential interactions by age, gender, race, previous hypertension, and left ventricular dysfunction using multivariable logistic regression in the National Heart Failure Project, a database of Medicare patients >65 years old recruited from 1998 through 2001. Among 56 942 patients, mean admission SBP was 147.0 ± 92.3 mmHg, 15% presenting with SBP >180 mmHg. Systolic blood pressure showed an inverse relationship with 30-day and 1-year mortality rates in all subgroups analysed. Using admission SBP of 120–149 mmHg as the reference, the adjusted odds ratios (95% confidence intervals) for 1-year mortality were 2.18 (1.77–2.69) for SBP <90 mmHg, 1.57 (1.47–1.69) for SBP 90–119 mmHg, 0.71 (0.67–0.76) for SBP 150–179 mmHg, 0.63 (0.57–0.68) for SBP 180–209 mmHg, and 0.51 (0.44–0.59) for SBP ≥ 210 mmHg.

Conclusion

Higher SBP on admission is associated with significant lower 30-day and 1-year mortality in patients hospitalized for HF. The relationship is strong, graded, independent of other clinical factors and consistent among subgroups.

Keywords

Systolic blood pressure • Heart failure • Mortality • Prediction

Introduction

Blood pressure is reported to have an inverse relationship with risk of mortality in patients with heart failure (HF). Although elevations in systolic blood pressure (SBP) are associated with an increased risk of developing HF¹ and antihypertensive treatment protects against this evolution to HF, more successfully so than against either stroke or myocardial infarction,^{2,3} among those hospitalized with HF, higher blood pressure at the time of presentation is associated with a lower risk of dying.^{4–8} Prior studies have described the association between high SBP

and lower mortality in these patients, exploring varying cut-offs predominantly ranging from 115 to 160 mmHg,^{5,9} and some of them with only a short-term follow-up.⁶ One study examined the continuous relationship between higher SBP levels and long-term mortality,⁸ although the consistency of this association among important subgroups of patients was not addressed. The different prognostic influence of high blood pressure according to age, gender, or race described by others^{10–12} emphasizes the importance of the study of the association between SBP and mortality in these different subgroups of patients hospitalized for HF.

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On the other hand, even if left ventricular dysfunction is not an important predictor of mortality in HF patients,^{4,5,8} most patients with normal left ventricular ejection fraction (LVEF) are hypertensive.¹³ However, the effect of the interaction between LVEF and blood pressure on prognosis has not been previously studied.

A better understanding of the association between blood pressure and mortality in a representative group of HF patients may improve risk stratification and generate questions about the underlying mechanism of this relationship and its therapeutic implications.

To address this issue, we investigated the relationship between the first blood pressure measurement and short- and long-term mortality in a nationally representative cohort of Medicare patients hospitalized for HF from 1998 through 2001 using data from the National Heart Failure (NHF) Project, an initiative funded by the Centres for Medicare & Medicaid Services (CMS) to improve the quality of care of Medicare beneficiaries hospitalized with cardiovascular diseases.¹⁴

Methods

The NHF project included the medical chart abstraction of almost 80 000 Medicare beneficiaries hospitalized with a principal discharge diagnosis of HF. Using these data, the project identified fee-for-service Medicare patients who were hospitalized with a principal discharge diagnosis of HF (International Classification of Diseases, Ninth Revision, Clinical Modification codes 402.01, 402.11, 402.91, 404.01, 404.91, or 428) between April 1998 and March 1999 or July 2000 and June 2001. The NHF project sample was restricted to single admissions of patients with valid Social Security numbers who were not receiving long-term haemodialysis and did not leave the hospital (transferred out or against medical advice) during treatment. For each time period, the project used a sampling strategy to randomly select up to 800 discharges in each state, sorted by age, race, gender, and treating hospital; states with <800 medical records were sampled in their entirety. The selected records were reviewed by trained data abstractors at central data abstraction centres. Medical records were abstracted for clinical data including medical history, comorbidity, findings at admission, in-hospital course, status at discharge, medication and procedure use, and laboratory and other diagnostic evaluations.

Study cohort

The NHF project produced an initial sample of 78 882 hospitalizations. Medical records meeting NHF exclusion criteria (chronic dialysis, no or invalid Social Security number, readmission) were excluded ($n = 4281$). We included patients aged 65 years and older who were hospitalized for HF, with the aim of evaluating the influence of SBP on 30-day and 1-year mortality. We excluded patients younger than 65 years ($n = 6558$), those who arrived by inter-hospital transfer ($n = 2419$) in which accurate admission information could not be identified, those without HF on admission ($n = 5003$), those with unknown SBP on admission ($n = 72$), those with severe comorbid diseases (acquired immune deficiency syndrome, chronic liver disease, leukaemia, lymphoma, immunological suppression or cancer) ($n = 6216$) that could strongly modify 1-year mortality risk, and those with unknown information about death ($n = 969$). Our final study sample had 56 942 patients.

Systolic blood pressure and other independent variables

The primary independent variable is SBP which was analysed continuously, then categorized into six groups (<90, 90–119, 120–149, 150–179, 180–209, and ≥ 210 mmHg) for risk estimation. Data on SBP reflect the first measurement obtained in the emergency department or, for patients directly admitted, what was first recorded in the medical record (including nursing notes, progress notes, graphics, and flow sheets). The first diastolic blood pressure (DBP) was also recorded and pulse pressure (PP) calculated as the difference between SBP and DBP.

Other variables included demographic characteristics (age, sex, race, pre-admission setting), non-cardiac comorbidities (mobility, continence, cerebrovascular accident, chronic pulmonary disease, dementia, diabetes, history of hypertension), characteristics on HF presentation (peripheral oedema, pulmonary oedema), other cardiac diseases (coronary artery bypass grafting, myocardial infarction, history of coronary artery disease, severity of left ventricular systolic dysfunction, and atrial fibrillation), laboratory data (serum sodium, albumin, glucose, haematocrit, and white blood cells), and medications (angiotensin-converting enzyme inhibitors, diuretics, angiotensin receptor blockers, digoxin, beta-blockers, and calcium channel blockers) at admission.

Documentation of LVEF was defined as a medical record notation of prior LVEF evaluation when reported, or in-hospital assessment of LVEF. Left ventricular ejection fraction categories were derived through information from current hospital assessment as well as previous LVEF evaluation. The classification of mild, moderate, and severe ventricular dysfunction was made according to the narrative description reported in the medical history. History of hypertension was considered present if there was any documentation by a physician that the patient had a previous hypertension diagnosis.

Outcomes

We determined patient mortality using the Medicare enrollment database, deriving mortality within 30 days and 1 year of admission from the admission date and the death date.

Statistical analysis

We reported data as mean $\pm$ standard deviation for continuous variables and number (percentage) for categorical variables. We categorized SBP at admission into groups as previously described and evaluated differences in patient characteristics among the SBP groups using the Pearson χ^2 test for categorical variables and analysis of variance for continuous variables.

We compared crude mortality within 30 days and 1 year of admission using the Pearson χ^2 test among the SBP groups overall and also stratified by patient age (<75, 75–84, and ≥ 85 years), gender (male or female), race (white, black, other), history of hypertension (no, yes), and LVEF (unknown, normal, mild, moderate, or severe). We evaluated the independent effect of SBP on mortality using multivariable logistic regression models in the overall cohort and also in the subgroups determined by age, gender, race, history of hypertension, and LVEF, adjusting for all clinical and laboratory variables described above. For SBP analyses, we considered the subgroup for SBP 120–149 mmHg, which includes normal blood pressure values, as the referent.

Owing to the sampling structure, we used survey analysis methods with the consideration of state-level weights and patient clustering within hospitals. We incorporated probability weights based on the inverse sampling fraction for each state in the analyses. We conducted all analyses using SAS 9.1 (SAS Institute Inc., Cary, NC, USA). The Yale

University School of Medicine Human Investigation Committee approved the use of the NHF database.

Results

Our study sample had a mean age of 79.7 ± 22.8 years; 59% were women and 85% were white. Baseline characteristics are shown in Table 1. Mean SBP was 147.0 ± 92.3 mmHg and mean DBP was 77.9 ± 55.1 mmHg. More than 50% of the patients had an SBP >140 mmHg, and $>15\%$ showed SBP values >180 mmHg. Overall, 30-day mortality was 9.8% and 1-year mortality was 37.5%.

Baseline patient characteristics differed across SBP groups (see Supplementary material online, Appendix). Patients in higher SBP ranges were more frequently female, younger, and healthier. With the exception of hypertension history, increasing SBP on admission was associated with fewer comorbidities and higher LVEF. Diastolic blood pressure was moderately correlated with SBP ($r = 0.63$).

Systolic blood pressure and mortality

Higher SBP on admission was associated with significantly lower 30-day and 1-year mortality (Table 2). This association persisted even at very high SBP levels (Figures 1 and 2). All levels of SBP above the reference were associated with a lower mortality risk at 30 days and 1 year. This effect was consistent across age, gender, race, history of hypertension, and LVEF subgroups (Table 3). There were no significant interactions between SBP and these groups except for LVEF ($P < 0.001$). The prognostic importance of SBP was higher for patients with more severe left ventricular dysfunction (Table 3).

In the multivariable analysis, higher SBP persisted as a powerful independent predictor of lower short-term and 1-year mortality in the entire spectrum of SBP after adjustment for confounders (Figure 3A). Patients with SBP ≥ 210 mmHg had an adjusted odds ratio for 1-year mortality of 0.51 (95% confidence interval 0.44–0.59) with respect to the 120–149 mmHg SBP range. The independent and inverse association between SBP and mortality was observed in all age, sex, and race subgroups as well as in patients with all degrees of LVEF (Table 3).

Diastolic blood pressure, pulse pressure, and mortality

Median DBP was 77 mmHg (interquartile range: 65–90 mmHg). Similar to SBP, higher DBP levels were associated with lower 30-day and 1-year mortality rates in unadjusted analysis that persisted after adjustment for baseline characteristics and SBP values (Figure 3B).

Median PP was 66 mmHg (interquartile range: 50–84 mmHg). Higher PP values were mostly driven by higher SBP levels than lower DBP, as both were correlated. Higher PP levels were associated with lower short- and long-term crude mortality rates. Contrary to the increasing benefit observed with higher levels of SBP and DBP, we did not find additional benefits in adjusted mortality over 120 mmHg (Figure 3C).

Table 1 Baseline characteristics

Demographics	
Age, mean (SD)	79.7 (22.8)
Female (%)	59.3
White (%)	85.1
Black (%)	11.8
Other (%)	3.1
Nursing facilities (%)	11.4
Non-cardiac comorbidities (%)	
Unable to walk independently	39.6
Incontinent	13.7
Chronic pulmonary disease	33.6
Diabetes	40.9
Creatinine, mean (SD)	1.5 (2.17)
Cardiac comorbidities (%)	
Coronary artery disease	59.5
Hypertension	65.6
CABG	23.8
PCI	10.0
Revascularization (PCI or CABG)	29.5
Myocardial infarction	29.5
Severity of left ventricular systolic dysfunction	
Unknown	34.8
Normal	24.4
Mild	9.6
Moderate	12.0
Severe	19.2
Left bundle branch block on ECG	15.4
Atrial fibrillation/flutter on ECG	30.1
Heart failure severity indicators	
Peripheral oedema (%)	73.1
Pulmonary oedema/heart failure on chest X-ray (%)	76.5
Sodium, mean (SD)	138.5 (14.3)
History of heart failure (%)	72.9
Other laboratory, mean (SD)	
Albumin (mg/dL)	3.4 (2.4)
Potassium (mEq/L)	4.3 (1.97)
Glucose (mg/dL)	152.9 (210)
White blood cell count, $n \cdot 10^3/\text{mm}^3$	9.1 (11)
Haematocrit (%)	36.8 (16.8)
Vital signs, mean (SD)	
Systolic blood pressure (mmHg)	147.0 (92.3)
Diastolic blood pressure (mmHg)	77.9 (55.1)
Pulse pressure (mmHg)	69.1 (72.5)
Heart rate (b.p.m.)	89.6 (65.1)
Respiratory rate	24.5 (19.5)
Medications on admission (%)	
Angiotensin-converting enzyme inhibitors	41.7
Angiotensin receptor blockers	9.2
Diuretics	74.3
Digoxin	39.6
Calcium-channel blockers	27.3
Beta-blockers	28.7
Aspirin	37.1

CABG, coronary artery bypass grafting; PCI, percutaneous coronary intervention.

Table 2 Crude outcomes (%) in different systolic blood pressure groups

	Systolic blood pressure (mmHg)						P-value
	<90	90–119	120–149	150–179	180–209	≥210	
30-Day mortality	34.1	18.6	10.1	5.7	4.4	2.9	<0.001
1-Year mortality	69.7	55.5	39.5	29.1	25.5	21.3	<0.001

Figure 1 Systolic blood pressure and 1-year mortality rates. **Figure 2** Kaplan–Meier crude survival curves for each subgroup of systolic blood pressure.

Discussion

This study demonstrates the independent and continuous relationship between higher first SBP measurement and lower short- and long-term mortality in older patients hospitalized for HF, with no upper threshold. This association is independent of patient clinical characteristics, is present across the entire spectrum of SBP even at the highest levels, and is consistent among groups of patients. Systolic blood pressure on admission is a strong prognostic factor, with implications for mortality risk, in a paradox that persists to very high levels of blood pressure.

The mechanisms of this association are not clear. Patients with higher SBP on admission are younger and healthier; however, baseline characteristics do not explain the better prognosis, as the effect on mortality after adjustment for these differences remains very strong. A second explanation may be related to differences in treatment patterns or responses. Physicians may be more aggressive using vasodilator therapies, e.g. angiotensin-converting enzyme inhibitors or beta-blockers, in patients with higher blood pressure levels. In addition, patients with higher SBP may better tolerate higher doses of these treatments than patients with lower levels of blood pressure. However, it is unlikely that a greater use or the use of higher doses explained the differences in short-term mortality, as the survival benefit of these drugs take months or years to be seen in randomized clinical trials. It is possible that the treatments used for patients with HF and low SBP increased mortality, as has been suggested for many inotropic agents.^{15–17} However, that would only partially explain the well-known higher mortality of patients with HF admitted with low SBP.^{5,8,18} The effect of treatments, such as beta-adrenergic receptor blockers and angiotensin-converting enzyme inhibitors, could differ across the SBP spectrum. This hypothesis cannot be ruled out because, although a number of trials have demonstrated a similar effect of the active drug independently of the level of SBP, most of these studies either used only two categories for comparison^{19,20} or excluded patients with very high SBP.^{20,21} It is possible that the better prognosis of the patients with highest SBP is partially due to their hospitalization having been caused by a hypertensive pulmonary oedema, rather than having been within the context of chronic HF with the full syndrome of other organ- and neurohormonal-associated changes.²² The existence of a distinct pathophysiology in patients with HF and higher blood pressure levels on admission seems also likely. The ability to raise blood pressure in the acute phase of HF regardless of LVEF may reflect a greater 'cardiovascular reserve' that can be mediated by a greater ability to achieve a vasoconstrictive response and/or a greater myocardial contractile reserve, translating into greater cardiac output. Thus, the capability of raising blood pressure during the acute phase of HF may be inversely associated with the severity of the disease, which is not well predicted by LVEF.

Previous studies have demonstrated the prognostic importance of SBP in HF patients.^{3–6,8} Some have mainly focused on lower levels of SBP as a predictor of higher mortality.^{5,18} Fonarow *et al.*⁵ reported that blood pressure above 115 mmHg was the best cutpoint to indicate lower risk. The association of high SBP and better prognosis has been described in patients with chronic stable HF^{23,24} and in patients requiring hospital admission for decompensation.^{4,6,25,26}

Table 3 One-year mortality stratified by systolic blood pressure group

Characteristics	Overall	Systolic blood pressure (mmHg)						P-value
		<90	90–119	120–149	150–179	180–210	>210	
Crude rate (%)								
Age (years)								
<75	27.7	60.0	46.0	29.6	19.2	16.1	14.0	<0.001
75 to <85	36.0	68.7	55.2	37.5	27.4	24.3	19.2	<0.001
≥85	49.2	81.1	65.6	51.6	41.4	36.3	34.7	<0.001
Race								
White	38.7	70.8	56.6	40.4	30.0	26.3	22.5	<0.001
Black	30.0	53.7	46.9	33.3	24.7	20.8	19.5	<0.001
Other	32.0	89.4	48.9	35.7	23.5	25.1	8.6	<0.001
Gender								
Male	40.3	72.0	58.2	40.9	29.1	25.8	17.5	<0.001
Female	35.5	67.1	52.7	38.4	29.1	25.3	22.9	<0.001
Hypertension								
No	42.6	77.5	56.9	42.1	31.4	27.9	21.2	<0.001
Yes	34.8	62.9	54.2	37.8	28.2	24.8	21.3	<0.001
Severity of LVSD								
Unknown	42.9	76.5	60.5	45.1	33.5	30.5	27.2	<0.001
Normal	28.4	44.6	45.0	30.6	24.4	21.0	20.6	<0.001
Mild	31.8	72.5	45.0	35.9	26.4	24.6	18.8	<0.001
Moderate	35.1	69.1	50.3	37.9	29.1	24.2	11.4	<0.001
Severe	43.5	73.4	59.7	41.4	29.9	25.7	16.4	<0.001
Adjusted OR (95% CI)								
Age (years)								
<75		1.99 (1.38–2.88)	1.60 (1.40–1.84)	1.00	0.64 (0.56–0.73)	0.57 (0.48–0.69)	0.49 (0.37–0.66)	
75 to <85		2.38 (1.73–3.27)	1.67 (1.50–1.87)	1.00	0.72 (0.65–0.79)	0.63 (0.55–0.72)	0.46 (0.36–0.59)	
≥85		2.36 (1.50–3.72)	1.49 (1.31–1.69)	1.00	0.74 (0.66–0.83)	0.64 (0.55–0.75)	0.60 (0.46–0.78)	
Race								
White		2.19 (1.74–2.74)	1.58 (1.47–1.70)	1.00	0.72 (0.67–0.77)	0.63 (0.57–0.69)	0.51 (0.43–0.61)	
Black		1.47 (0.85–2.52)	1.47 (1.16–1.86)	1.00	0.73 (0.60–0.89)	0.68 (0.53–0.86)	0.62 (0.44–0.88)	
Other		17.51 (4.07–75.29)	1.42 (0.89–2.26)	1.00	0.58 (0.38–0.89)	0.71 (0.40–1.24)	0.25 (0.08–0.72)	

Gender						
Male	2.18 (1.61–2.96)	1.63 (1.47–1.80)	1.00	0.69 (0.63–0.77)	0.64 (0.55–0.74)	0.41 (0.30–0.56)
Female	2.08 (1.56–2.79)	1.48 (1.35–1.64)	1.00	0.74 (0.68–0.80)	0.64 (0.58–0.72)	0.58 (0.49–0.69)
Hypertension						
No	2.81 (2.04–3.86)	1.52 (1.37–1.68)	1.00	0.71 (0.63–0.79)	0.65 (0.54–0.77)	0.48 (0.32–0.70)
Yes	1.79 (1.36–2.36)	1.59 (1.44–1.74)	1.00	0.73 (0.67–0.79)	0.64 (0.58–0.72)	0.54 (0.46–0.64)
Severity of LVSD						
Unknown	2.62 (1.87–3.67)	1.62 (1.43–1.83)	1.00	0.69 (0.62–0.77)	0.65 (0.57–0.76)	0.55 (0.43–0.70)
Normal	0.99 (0.60–1.62)	1.55 (1.31–1.83)	1.00	0.86 (0.75–0.98)	0.75 (0.62–0.90)	0.74 (0.56–0.97)
Mild	3.73 (1.61–8.65)	1.18 (0.91–1.53)	1.00	0.66 (0.53–0.82)	0.63 (0.47–0.83)	0.49 (0.31–0.77)
Moderate	2.27 (1.19–4.31)	1.29 (1.04–1.59)	1.00	0.70 (0.58–0.85)	0.57 (0.43–0.74)	0.28 (0.15–0.50)
Severe	2.45 (1.65–3.65)	1.69 (1.47–1.94)	1.00	0.70 (0.60–0.82)	0.58 (0.45–0.75)	0.32 (0.18–0.56)

CI, confidence interval; LVSD, left ventricular systolic dysfunction; OR, odds ratio.

However, there are a number of differences between the current study and previous studies. Many previous studies included a small number of selected patients,²⁶ did not adequately examine higher SBP levels,²⁶ or were focused on short-term mortality.^{6,27} Our study of a representative population confirms previous findings and demonstrates the association of higher SBP levels and lower mortality across the full spectrum of SBP values, in short- and long-term follow-up, and includes information about the consistency of the association across different patient subgroups. Even if the number of cases in each subgroup is not very high, our sample contained 2176 patients with SBP of 210 mmHg or above, which provides a reasonable number of cases in each subgroup with which to make comparisons.

Diastolic blood pressure, pulse pressure, and mortality

Few studies have evaluated the relationship between DBP and mortality in HF patients. Lee *et al.*²⁴ demonstrated that DBP < 60 mmHg increased mortality in chronic HF patients with left ventricular dysfunction. When they analysed the relationship, considering DBP as a continuous variable, the association between higher DBP and lower mortality was evident, but the upper DBP levels in their study population were lower than those in our population. Similarly, the OPTIMIZE-HF registry found a decrease in hospital mortality per each 10 mmHg of DBP increase up to 100 mmHg.²⁷ We have shown that even higher levels of DBP are associated with a better prognosis.

Pulse pressure at admission is also a strong independent predictor of better survival in patients hospitalized with HF in our study. Elevated PP relates to arterial stiffness and is associated with greater cardiovascular events and mortality risk. In older patients, PP seems to be a better predictor of HF development than SBP and DBP.²⁸ However, in patients with HF, it has been speculated that the better prognosis observed in patients with higher PP values may be due to a greater inotropic reserve.²⁵

The results of this study should be evaluated in the context of its limitations. The analysis was restricted to fee-for-service Medicare patients—the predominate form of government coverage for the elderly in the USA in which doctors are paid for each service they provide—aged 65 years or older and hospitalized for HF, and the findings may not be applicable to other populations. Nevertheless, the majority of HF patients are aged 65 years or older, and the proportion of patients included in the Medicare managed care system is very low.

The retrospective analysis of the results of a database is also associated with other limitations, which theoretically might have had an influence on the findings of this study, such as the risk of HF 'upcoding',²⁹ the use of only first blood pressure measurement, or the lack of standardized blood pressure measurement, including time of the day, person who measured it or relationship between the taking of medications and timing of BP recordings. However, the large number of cases, and the remarkable consistency of the findings across subgroups as well as the exclusion of patients who did not have a diagnosis of HF at the time of admission makes it unlikely that these limitations played an important role in the results. Finally, the hypothesis that patients with higher

Figure 3 Adjusted odds ratios and 95% confidence intervals for 1-year mortality rate for systolic blood pressure (A), diastolic blood pressure (B), and pulse pressure (C) groups. Reference value = 120–149 mmHg for SBP and 60–89 mmHg for diastolic and pulse pressures.

SBP at admission received different treatments, agents or doses of vasodilators during follow-up, cannot be discounted as noted.

In conclusion, our study enriches the profiling of admission SBP as a risk factor, raises the issue of an underlying pathophysiological mechanism contributing to better prognosis of high blood pressure in HF patients and, more importantly, the issue of potential therapeutic implications. Whether the response to the recommended therapies (i.e. angiotensin-converting enzyme inhibitors or beta-blockers) differs in patients with very high blood pressure levels, and if the doses and titration should vary accordingly, merit further research.

Supplementary material

Supplementary material is available at *European Journal of Heart Failure* online.

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