



Original article

Systolic blood pressure on admission as a marker of fluid re-distribution in acute heart failure



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ABSTRACT

Background: The relation between systolic blood pressure (sBP) on admission and the extent of fluid re-distribution in patients with acute heart failure (AHF) remains unclear. This study aimed to investigate this relation.

Methods: We enrolled consecutive patients who were admitted for AHF in our cardiology department and divided them into three groups according to the tertiles of sBP on admission as follows: low, intermediate, and high sBP groups. Weight changes and estimated relative plasma volume changes (Δ ePV) on admission and 24 h later were determined in each patient. Δ ePV were calculated with the Strauss formula using hemoglobin and hematocrit levels. Univariate and multiple regression analyses were performed to investigate the relation between sBP and Δ ePV.

Results: The Δ ePV of low, intermediate, and high sBP groups were 104.3%, 108.2%, and 121.3%, respectively. High sBP group showed a significantly larger Δ ePV than the other two groups ($p < 0.001$ and 0.004 , respectively). The body weight of patients in the high sBP group slightly but significantly decreased within 24 h (-0.64 ± 0.92 kg, $p = 0.002$). The initial sBP had a significant correlation with Δ ePV (R^2 , 0.295; $p < 0.001$). Multiple regression analysis showed significant association between initial sBP with Δ ePV.

Conclusions: These results indicate that large amounts of extravasated fluid existed on admission in patients with a high initial sBP. The sBP on admission could be a simple and useful indicator for the extent of fluid re-distribution in AHF.

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Introduction

Fluid accumulation has been thought to be an important aspect of acute heart failure (AHF). Previous studies have shown that fluid extravasation and intravascular dehydration are observed in acute cardiogenic pulmonary edema [1–3]. In addition to fluid accumulation, fluid re-distribution has recently been pointed out as another pathophysiological aspect in AHF syndrome [4–6].

Plasma volume has been conventionally evaluated using radioiodinated human serum albumin [2]. Instead of using the radio-

isotope method, relative blood volume change could have been measured using the hemoglobin (Hb) concentration or hematocrit (Hct) in previous studies [3,7–9]. The estimated relative plasma volume change (Δ ePV) which is calculated using the Strauss formula along with Hb and Hct levels, has also been used in heart failure patients [10–13].

It has been proposed that low systolic blood pressure (sBP) indicates fluid accumulation in the majority of patients with AHF, whereas a high sBP indicates plasma extravasation and intravascular dehydration without confirmation [14]. This idea of the relation between the initial sBP and fluid re-distribution has influenced the therapeutic strategies in patients with AHF, particularly those in the clinical scenarios [14]. However, the relation between the initial sBP and fluid re-distribution in patients with AHF has not been precisely elucidated.

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By measuring ΔePV calculated using Hb and Hct levels, this study aimed to investigate the importance of initial sBP in terms of predicting a pathophysiological fluid shift in patients with AHF.

Methods

We performed a retrospective, observational, and single center study.

Consecutive patients who were admitted to the Cardiology Department at the Japanese Red Cross Medical Center for AHF diagnosed using the Framingham criteria of congestive heart failure [15] were enrolled from April 2016 to June 2017. Exclusion criteria were as follows: (1) lack of laboratory data on admission or within 48 h from admission, (2) cardiac arrest before or on arrival at the hospital, (3) initial sBP too low to measure, (4) use of intra-aortic balloon pumping (IABP) or veno-arterial extracorporeal membrane oxygenation (VA-ECMO), and (5) active bleeding or need for blood transfusion. Patients were divided into three groups according to the tertiles of initial sBP on admission: low, intermediate, and high sBP groups.

The following factors were included for evaluation: age, sex, body weight, New York Heart Association (NYHA) functional classification, subjective symptoms, heart rate, sBP, SpO_2 , laboratory data, and medications from medical records. The sBP, Hb, Hct, and body weight on admission and 24 h later were used. Laboratory data at 24 h post-admission or data nearest from post-admission to 48 h later were used. The differences in the following parameters between admission and 24 h after admission were used: sBP, Hb, Hct, and body weight (ΔsBP , ΔHb , ΔHct , and ΔBW , respectively). We checked chest radiographs on admission to determine the presence of pulmonary edema and pleural effusion. Data from echocardiograms that were performed at the nearest time point from admission were used. Echocardiogram parameters used include left ventricular end-diastolic diameter (LVDd), left ventricular end-systolic diameter (LVDs), ejection fraction (EF), and inferior vena cava (IVC) diameter. IVC collapsibility index (IVC-CI) was defined as $100 \times (\text{maximum IVC} - \text{minimum IVC}) / \text{maximum IVC}$. To estimate the relative plasma volume change between admission and 24 h later (ΔePV), the Strauss formula [10] was used as follows:

$$\Delta\text{ePV} = 100 \times \frac{\text{Hemoglobin (admission)}}{\text{Hemoglobin (24h later)}} \times \frac{100 - \text{Hematocrit (24h later)}}{100 - \text{Hematocrit (admission)}}$$

Continuous variables are presented as means \pm standard deviation, whereas categorical variables are presented as counts. Normality was judged using the Shapiro–Wilk test, whereas equality of variance was assessed using the Levene test. In three-group analyses, we compared the continuous variables that had normality and equal variances using one-way analysis of variance. The Welch test was performed to compare the continuous variables that had normality, but whose variances were not equal. Factors that were not normally distributed were compared using the Kruskal–Wallis test. The chi-square or Fisher's exact test were appropriately used to compare proportions of categorical variables between the groups. In two group analyses, the *t*-test was used to compare the continuous variables having normality, whereas the Mann–Whitney test was used to compare those variables whose variances were not equal. Paired *t*-test or Wilcoxon signed-rank test was appropriately performed to compare parameters that were repeatedly measured, such as body weight. Spearman's rank correlation test was used to confirm the correlation of parameters. We performed simple linear regression analysis to examine the association between ΔePV and other variables. To determine clinical parameters that were associated with ΔePV , multiple regression analysis was performed with a stepwise method using the factors with $p < 0.05$, except those causing multicollinearity. A value of $p < 0.05$ was considered statistically significant. All analyses were performed using IBM SPSS Statistics version 23 (Armonk, NY, USA). This study was approved by the institutional review board of the Japanese Red Cross Medical Center.

Results

We enrolled 134 consecutive patients with AHF from April 2016 to June 2017. A total of 40 patients were excluded according to the exclusion criteria as follows: lack of laboratory data on admission or within 48 h from admission ($n = 20$), cardiac arrest before arriving to hospital ($n = 2$), initial sBP too low to measure ($n = 1$), use of IABP or VA-ECMO ($n = 3$), and active bleeding or need for blood transfusion ($n = 14$). A total of 94 patients were included for the analysis (Fig. 1). The second measurements of Hb and Hct

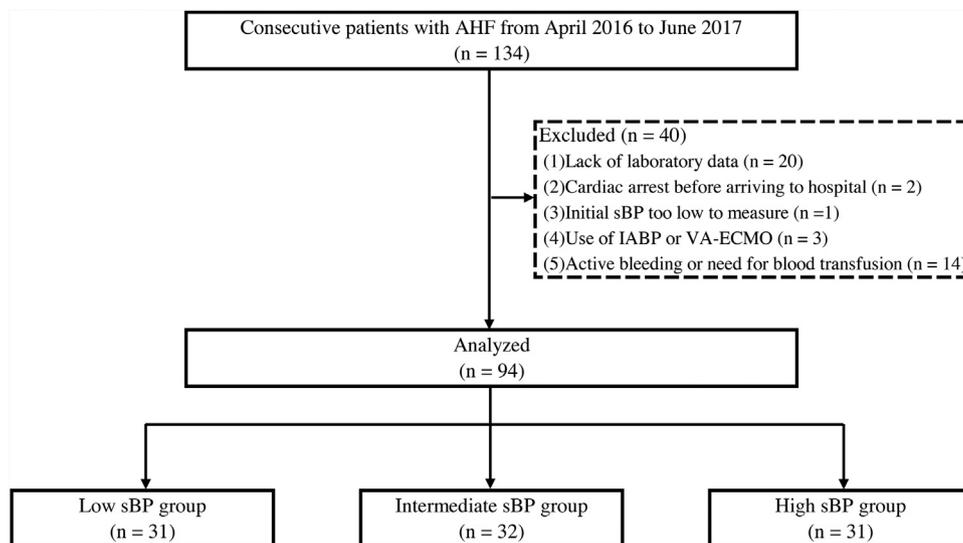


Fig. 1. Study flow chart. Consecutive 134 patients admitted to the Cardiology Department at the Japanese Red Cross Medical Center for AHF from April 2016 to June 2017 were enrolled. A total of 40 patients were excluded by the criteria, and finally, 94 patients were analyzed.

AHF, acute heart failure; IABP, intra-aortic balloon pumping; sBP, systolic blood pressure; VA-ECMO, veno-arterial extracorporeal membrane oxygenation.

levels were performed at 23 h 47 min \pm 10 h 25 min after admission (mean \pm SD, range from 6 h 37 min to 46 h 46 min).

Table 1 shows patient characteristics. The initial sBP was significantly higher in the high sBP group (184.4 \pm 24.4 mmHg) as compared to the other groups. The high sBP group had the highest Hb concentration and Hct on admission (12.6 \pm 2.2 g/dL and 38.1 \pm 6.1, respectively), whereas the low sBP group had the lowest (11.2 \pm 1.5 g/dL and 33.9 \pm 4.3, respectively). The high sBP group had higher NYHA classification score (3.6 \pm 0.7) and needed more frequent positive pressure ventilation (38.7%). Nitrates, carperitide, and calcium channel blockers were more frequently used in the high sBP group (Table 1).

Fig. 2 shows differences in sBP, body weight, Hb, and Hct between admission and 24 h later. The sBP, Hb, and Hct significantly decreased more prominently in the high sBP group than in the low and intermediate sBP groups (Fig. 2A, C, and D). The amount of body weight significantly decreased in all groups (low, intermediate, and high sBP groups; $p < 0.001$, $p < 0.001$, $p = 0.002$, respectively), and the extent of body weight loss in the three groups was not significantly different ($p = 0.058$; Fig. 2B). Δ ePV was significantly higher in the high sBP group than in the low and intermediate sBP groups ($p < 0.001$ and $p = 0.004$, respectively; Fig. 3).

Table 1
Patient characteristics.

	Low sBP group n = 31	Intermediate sBP group n = 32	High sBP group n = 31	p-value
Age (years)	78.9 \pm 8.8	73.4 \pm 18.6	80.8 \pm 9.8	0.165
Male	48.4	68.8	64.5	0.220
NYHA functional classification	3.3 \pm 0.8	3.0 \pm 0.9	3.6 \pm 0.7	0.004
Etiology				
Ischemic heart disease	41.9	25.0	38.7	0.325
Cardiomyopathy	19.4	15.6	29.0	0.408
Valvular heart disease	19.4	21.9	11.3	0.636
Bradyarrhythmia	6.5	6.3	6.5	0.500
Tachyarrhythmia	3.2	12.5	4.8	0.247
Others	9.7	18.8	9.7	0.290
Medications prior to admission				
β blockers	51.6	43.8	35.5	0.440
Renin-angiotensin system inhibitors	51.6	53.1	35.5	0.302
Mineralocorticoid receptor antagonists	32.3	21.9	3.2	0.013
Calcium channel blockers	22.6	31.3	16.1	0.363
Loop diuretics	74.2	59.4	22.6	<0.001
Inotropic agents	19.4	21.9	6.5	0.101
Observations at admission				
Dyspnea	73.3	87.5	93.5	0.031
Edema	76.7	78.1	61.3	0.305
Body weight (kg)	58.3 \pm 13.3	62.7 \pm 15.9	61.5 \pm 18.2	0.706
Heart rate (beats per minute)	92.9 \pm 23.8	90.3 \pm 23.9	104.3 \pm 27.9	0.071
Initial systolic blood pressure (mmHg)	104.7 \pm 12.0	133.2 \pm 7.4	184.4 \pm 24.4	<0.001
AST (U/L)	45.7 \pm 31.2	43.9 \pm 33.1	52.7 \pm 51.2	0.384
ALT (U/L)	37.5 \pm 37.4	31.7 \pm 33.3	45.2 \pm 48.5	0.316
TP (g/dL)	6.7 \pm 0.6	6.7 \pm 0.8	6.9 \pm 0.7	0.479
ALB (g/dL)	3.4 \pm 0.4	3.4 \pm 0.3	3.6 \pm 0.5	0.037
BUN (mg/dL)	32.5 \pm 18.4	26.6 \pm 13.2	24.9 \pm 9.3	0.185
Cre (mg/dL)	1.41 \pm 0.94	1.24 \pm 0.68	1.02 \pm 0.51	0.139
BUN/Cre	25.3 \pm 7.2	22.6 \pm 6.7	28.9 \pm 17.8	0.246
eGFR (ml/min/1.73 m ²)	45.1 \pm 23.1	52.6 \pm 23.3	59.0 \pm 24.8	0.072
UA (mg/dL)	7.6 \pm 2.0	7.3 \pm 2.2	5.7 \pm 1.2	<0.001
Na (mEq/L)	137.6 \pm 4.3	138.2 \pm 3.3	139.6 \pm 3.1	0.267
K (mEq/L)	4.5 \pm 0.6	4.4 \pm 0.7	4.3 \pm 0.7	0.439
BNP (pg/ml)	1727 \pm 2477	908 \pm 920	976 \pm 747	0.142
WBC (/ μ L)	6865 \pm 2269	7252 \pm 2452	9694 \pm 3326	<0.001
Hb (g/dL)	11.2 \pm 1.5	12.1 \pm 2.3	12.6 \pm 2.2	0.019
Hct (%)	33.9 \pm 4.3	36.6 \pm 6.6	38.1 \pm 6.1	0.013
Pulmonary edema	19.4	9.4	61.3	<0.001
Pleural effusion	80.6	68.8	61.3	0.527
LVDd (mm)	58.4 \pm 11.4	59.5 \pm 11.7	56.0 \pm 9.6	0.456
LVDs (mm)	46.1 \pm 14.3	46.1 \pm 14.2	42.6 \pm 11.4	0.482
EF (%)	41.0 \pm 18.7	45.7 \pm 18.3	47.5 \pm 16.4	0.252
IVC (maximum, mm)	19.0 \pm 4.4	22.0 \pm 6.2	17.3 \pm 4.6	0.002
IVC (minimum, mm)	10.8 \pm 5.2	13.2 \pm 7.3	8.6 \pm 5.8	0.014
IVC collapsibility index (%)	44.4 \pm 17.2	43.5 \pm 21.8	53.6 \pm 19.4	0.096
Treatments after admission				
Positive pressure ventilation	9.6	3.1	38.7	<0.001
Diuretics	90.3	100.0	90.3	0.093
Nitrates	12.9	9.4	58.1	<0.001
Carperitide	0.0	9.4	22.6	0.006
Inotropic agents	32.3	25.0	9.7	0.093
Calcium channel blockers	12.9	18.8	38.7	0.042

Values are expressed as mean \pm standard deviation or percentage unless otherwise indicated.

ALB, albumin; ALT, alanine aminotransferase; AST, aspartate aminotransferase; BNP, brain natriuretic peptide; BUN, blood urea nitrogen; Cre, creatinine; EF, ejection fraction; eGFR, estimated glomerular filtration rate; Hb, hemoglobin; Hct, hematocrit; IVC, inferior vena cava; LVDd, left ventricular end-diastolic diameter; LVDs, left ventricular end-systolic diameter; NYHA, New York Heart Association; sBP, systolic blood pressure; TP, total protein; UA, uric acid; WBC, white blood cell.

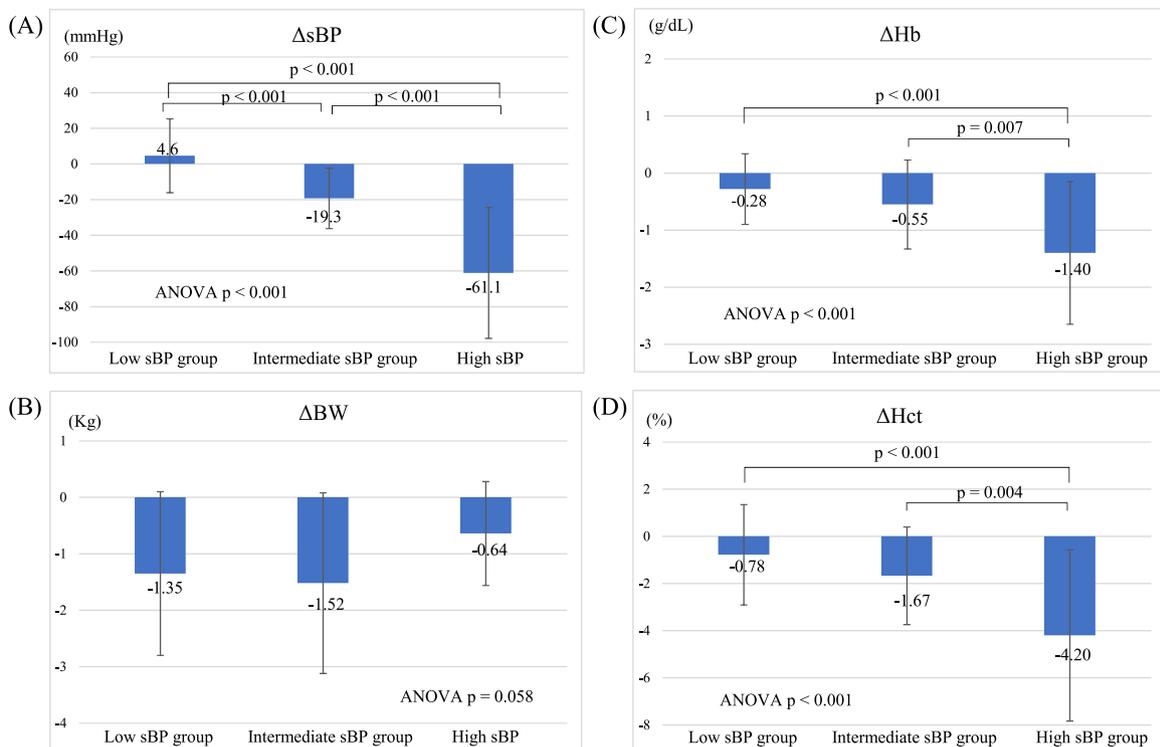


Fig. 2. Changes in systolic blood pressure (A), body weight (B), hemoglobin (C), and hematocrit (D) between admission and 24 h later in three groups. (A) The high sBP group showed significantly bigger change in Δ sBP than the low and intermediate sBP groups (both $p < 0.001$). (B) Body weight significantly decreased in all groups (low, intermediate, and high sBP group: $p < 0.001$, $p < 0.001$, and $p = 0.002$, respectively). The extent of body weight loss in the three groups was not statistically significant ($p = 0.058$). (C) The high sBP group showed significantly bigger change in Δ Hb than the low and intermediate sBP groups ($p < 0.001$ and $p = 0.007$, respectively). (D) The high sBP group showed significantly bigger change in Δ Hct than the low and intermediate sBP groups ($p < 0.001$ and $p = 0.004$, respectively). Δ sBP, change in systolic blood pressure at admission and 24 h later; Δ BW, change in body weight at admission and 24 h later; Δ Hb, change in hemoglobin concentration at admission and 24 h later; Δ Hct, change in hematocrit level at admission and 24 h later; ANOVA, analysis of variance.

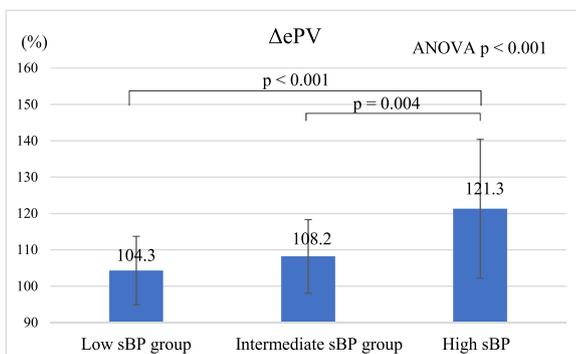


Fig. 3. Δ ePV in the three groups. The high sBP group showed significantly higher Δ ePV than the low and intermediate groups ($p < 0.001$, $p = 0.004$, respectively). Δ ePV, change in estimated relative plasma volume at admission and 24 h later; ANOVA, analysis of variance; sBP, systolic blood pressure.

Univariate analysis showed that the following factors had a significant correlation with Δ ePV: NYHA functional classification, dyspnea, heart rate, initial sBP, Δ sBP, Δ BW, total protein, albumin, initial Hb, Hct, pulmonary edema, use of positive pressure ventilation, nitrates, and calcium channel blockers after admission (Table 2).

To determine the clinical parameters that were associated with Δ ePV, multiple regression analysis was performed for the factors with $p < 0.05$ in the univariate analysis. For factors that were significant by univariate analysis, Δ sBP had multicollinearity with the initial sBP ($\rho = -0.817$ and $p < 0.001$), and the initial Hct also had multicollinearity with Hb ($\rho = 0.982$ and $p < 0.001$). Because of

these multicollinearities, we excluded Δ sBP and initial Hct from the multiple regression analysis. The initial sBP, use of nitrates, initial total protein and Hb concentrations, and Δ BW showed significant association with Δ ePV (Table 3). The initial sBP had a significant positive correlation with Δ ePV ($R^2 = 0.295$ and $p < 0.001$; Fig. 4).

Discussion

The present study showed that patients with high sBP had higher initial Hb and Hct than those with low and intermediate sBP (Table 1). Patients in the high sBP group had significantly decreased sBP, Hb, and Hct and significantly increased estimated plasma volumes after the start of treatment (Figs. 2 and 3). These results reveal that in patients with a higher initial sBP on admission, the initial plasma volume was low compared to other patients, then this plasma volume increased after the start of treatments which resulted in decrease of Hb and Hct levels. Our results may suggest that more extravasating fluid returned to the intravascular space in patients with a higher initial sBP. In fact, their body weight decreased significantly despite an increase in plasma volume (Fig. 2B and 3).

Plasma volume can be changed by several factors including bleeding and water intake or loss from the body. Additionally, fluid shift between the intravascular and extravascular spaces also causes plasma volume change. Water intake and loss could influence the body weight of patients, but fluid shift theoretically does not influence body weight. In this study, the estimated plasma volume increased in all groups (Fig. 3), whereas body weight significantly decreased in all groups at 24 h after the start of

Table 2
Predictors of ΔePV on univariate regression analyses.

	B	SE B	β	p-value
Age (years)	0.035	0.118	0.030	0.771
Male	-2.103	3.232	-0.068	0.517
NYHA functional classification	4.137	1.970	0.218	0.039
Dyspnea	9.032	4.217	0.218	0.035
Edema	-5.277	3.454	-0.157	0.130
Heart rate (beats per minute)	0.233	0.057	0.392	<0.001
Initial systolic blood pressure (mmHg)	0.226	0.036	0.543	<0.001
Δ Systolic blood pressure (mmHg)	-0.231	0.035	-0.568	<0.001
Δ Body weight (kg)	3.456	1.306	0.298	0.010
TP (g/dL)	5.898	2.208	0.268	0.009
ALB (g/dL)	11.944	3.466	0.338	0.001
BUN/Cre	-0.106	0.134	-0.082	0.430
UA (mg/dL)	-1.147	0.779	-0.152	0.144
BNP (pg/ml)	7.087×10^{-5}	0.001	0.007	0.943
Hb (g/dL)	2.770	0.704	0.381	<0.001
Hct (%)	0.985	0.246	0.385	<0.001
Pulmonary edema	7.897	3.361	0.238	0.021
Pleural effusion	-1.298	3.579	-0.038	0.718
EF (%)	0.026	0.093	0.030	0.777
IVC collapsibility index (%)	-4.193	8.313	-0.054	0.615
Positive pressure ventilation	13.626	3.979	0.338	0.001
Diuretics	-3.468	6.464	-0.056	0.593
Nitrates	16.22	3.157	0.472	<0.001
Carperitide	6.027	5.094	0.122	0.240
Inotropic agents	-4.089	3.775	-0.112	0.282
Calcium channel blockers	10.163	3.584	0.283	0.006

ΔePV , estimated relative plasma volume change between admission and 24 h later; β , standardized coefficient; ALB, albumin; B, unstandardized coefficient; BNP, brain natriuretic peptide; BUN, blood urea nitrogen; Cre, creatinine; EF, ejection fraction; Hb, hemoglobin; Hct, hematocrit; IVC, inferior vena cava; NYHA, New York Heart Association; SE B, the standard error for the unstandardized coefficient; TP, total protein; UA, uric acid.

Table 3
Predictors of ΔePV on multivariate regression analysis with stepwise method.

	B	SE B	β	p-value
Initial systolic blood pressure (mmHg)	0.121	0.050	0.276	0.017
Hb (g/dL)	1.825	0.655	0.254	0.007
Nitrates	10.985	3.792	0.301	0.005
TP (g/dL)	5.555	2.012	0.235	0.007
Δ Body Weight (kg)	2.067	1.020	0.176	0.047

$R^2 = 0.537$, ANOVA $p < 0.001$.

ΔePV , estimated relative plasma volume change at admission and 24 h later; β , standardized coefficient; ANOVA, analysis of variance; B, unstandardized coefficient; Hb, hemoglobin; R, multiple correlation coefficient; SE B, the standard error for the unstandardized coefficient; TP, total protein.

treatment, including diuretics (Fig. 2B). These results indicate that the increase in plasma volume could be attributed to the fluid shift from the extravascular to the intravascular spaces rather than water intake and the extent of fluid shift was more prominent in the high sBP group. In fact, ΔePV in the high sBP group was the largest among the three groups (Fig. 3), whereas their body weight decreased (Fig. 2B).

Our results indicated that the high sBP group had more extravasated fluid (Figs. 2–4) and relative intravascular dehydration as indicated by high initial Hb and Hct levels compared to other groups before the start of treatments (Table 1). The initial sBP could be a useful parameter because sBP had significant correlation with ΔePV (Table 3 and Fig. 4). We could assume from the results that a pathophysiological aspect of the high sBP group may be fluid re-distribution rather than fluid accumulation. The present and previous studies [1–3,14,16,17] support the previously proposed pathophysiological consideration of clinical scenario 1, i.e. patients with AHF whose initial sBP is high are often systemically euvoletic or hypovolemic, and their pulmonary edema is caused by fluid

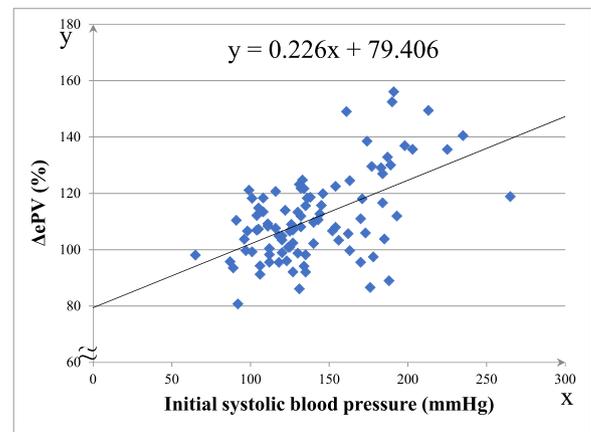


Fig. 4. Simple linear regression analysis between initial systolic blood pressure and ΔePV . Initial systolic blood pressure showed significant positive correlation with ΔePV ($R^2 = 0.295$, $p < 0.001$). ΔePV , change in estimated relative plasma volume at admission and 24 h later; R, multiple correlation coefficient.

extravasation secondary to an acute elevation of filling pressure, which parallels the increase in systemic blood pressure [14]. Previous studies also demonstrated that AHF can occasionally occur without significant excess fluid accumulation [18–20]. Thus, fluid re-distribution instead of fluid accumulation could be the main pathophysiological status in AHF patients with higher sBP. Our results could confirm this hypothesis at least in part.

A previous study revealed that nitrates diminish peripheral vascular resistance and venous tone as well as decrease left ventricular end-diastolic pressure (LVEDP) in heart failure [21]. Our results show that the use of nitrates also had a significant correlation with ΔePV (Table 3). We speculate that in our case, nitrates made fluid return from the extravascular space into the intravascular compartment possibly through a decrease in LVEDP as previously reported [22,23]. This suggests that nitrates could be a useful intervention in the return of extravasated fluid to the intravascular space. Another previous study reported that vasodilators show more beneficial effects than diuretics in the improvement of lung congestion and the prognosis of patients with AHF who have severe pulmonary edema [24]. Administration of nitrates in patients with a high sBP and AHF is reasonable from the aspect that in these patients, fluid shift from the intravascular system to the extravascular space occurs and nitrates could improve it.

To the best of our knowledge, this is the first study that shows the relation between sBP on admission and the extent of fluid re-distribution in AHF. We speculate that our results can explain the Hb concentration decrease after the start of treatment in patients with AHF whose initial sBP is relatively high. However, it is important to eliminate active bleeding, which can also cause a decrease in Hb.

A previous study reported that IVC-CI on admission could be a marker of fluid shift in patients with AHF [7]. In contrast, our results did not show a correlation between IVC-CI and ΔePV . We speculate that the reason for these conflicting results may be the timing of the measurement of IVC-CI. Asahi et al. [7] measured IVC-CI on admission, but we adopted the value that was measured at the nearest point from admission. In many cases, the measurement of IVC-CI was performed after the start of treatment.

We calculated ΔePV using the Strauss formula [10], but we did not measure the exact plasma volume change using radio-isotope methods. The gold standard for measuring plasma volume is the radio-isotope method, but radiation exposure is a burden to patients. Conversely, the Strauss formula only consists of the Hb

concentration and Hct [10] and is easy to calculate. Both values are included in routine blood tests and, the plasma volume can be calculated using the formula in routine practice. A previous study showed that Δ ePV calculated using the Strauss formula has a prognostic significance in heart failure [12,13]. Thus, we used this formula to estimate the relative plasma volume change.

This study has some limitations. Firstly, this was a single-center study, and the sample size was small. Almost all patients were Japanese. Therefore, racial difference cannot be eliminated. Secondly, we did not exclude patients who received treatments for anemia other than blood transfusion. Some patients were administered iron, erythropoiesis-stimulating agents, and vitamin B12, but we considered that these medications could not significantly influence our results because the production of red blood cells after the initiation of these treatment agents was time consuming. Thirdly, we used body weight as a representative value of water balance. We could not check the amount of drinking water or meal consumed, intravenous infusion provided, urine excreted, stools, insensible water loss, and the blood samples. Thus, we were not able to check the exact water balance. Finally, we estimated the relative plasma volume change using the Strauss formula, but we did not measure the exact plasma volume using radio-isotope methods.

Conclusions

AHF patients with a high sBP on admission showed a marked increase in the plasma volume after the start of treatment, despite their weight loss. This plasma volume expansion could be attributed to fluid return from the extravascular space to the intravascular space. Initial sBP significantly correlated with the increase in the plasma volume, as revealed by univariate and multiple regression analyses. These results indicated that large amounts of extravasated fluid existed on admission in patients with a high initial sBP and that treatment made the fluid to shift into the intravascular space. The sBP on admission could be a simple and useful indicator of fluid re-distribution in AHF.

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None.

Conflict of interest

None.

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