Published in final edited form as:

J Invasive Cardiol. 2018 February ; 30(2): 56–61.

Predictors of Hemodynamic Improvement and Stabilization Following Intraaortic Balloon Pump Implantation in Patients With Advanced Heart Failure

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Abstract

Objectives—The intraaortic balloon pump (IABP) is currently an essential tool to improve hemodynamics in patients with advanced heart failure (HF). This study investigated predictors for hemodynamic improvement or stabilization with IABP therapy in patients with advanced HF.

Methods—Patients with advanced HF and hemodynamic deterioration treated with IABP were enrolled in this retrospective study. Invasive hemodynamics were measured before IABP implantation and 2 weeks after IABP initiation. Significant degree of hemodynamic improvement was defined as 30% improvement in all three of the following variables: central venous pressure (CVP); pulmonary capillary wedge pressure (PCWP); and cardiac index (CI). Hemodynamic stabilization was counted in patients reaching CVP <12 mm Hg, PCWP <18 mm Hg, and CI >2.0 L/min/m^2 or CI >2.2 L/min/m^2 on inotropes.

Results—Ninety-one patients (55 ± 12 years; 78% males) were evaluated. Seventeen patients (18.7%) achieved significant hemodynamic improvement, and baseline CVP >16 mm Hg was associated with this endpoint (P<.05). Thirty-two patients (35.2%) achieved hemodynamic stabilization; lower baseline heart rate (HR) and PCWP were associated with this stabilization (P<.05). Patients with HR <92 beats/min and PCWP <25 mm Hg achieved hemodynamic stabilization more frequently than those without HR <92 beats/min and PCWP <25 mm Hg (66.7% vs 19.7%; P<.05).

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Disclosure: The authors have completed and returned the ICMJE Form for Disclosure of Potential Conflicts of Interest. Dr Imamura reports funding from the Postdoctoral Fellowship for Research Abroad of Japan Society for the Promotion of Science. The remaining authors report no conflicts of interest regarding the content herein.

Conclusion—Elevated CVP and lower HR and PCWP before IABP initiation help predict high response to IABP.

Keywords

heart failure; unloading; inotropes; counterpulsation; IABP; CVP

Intraaortic balloon pump (IABP) has been an essential tool to support hemodynamics in patients with cardiogenic shock following acute myocardial infarction or in the postcardiotomy setting.[1–3] However, recent randomized control studies and meta-analyses do not show survival benefit of IABP use in these settings.[4–6]

In contrast, studies evaluating the efficacy of IABP support in patients with advanced heart failure (HF) are lacking. In a small population study, IABP support was effective in patients with non-ischemic cardiomyopathy as well as those with ischemic etiology.[7] Furthermore, prolonged IABP support in patients with advanced HF improved hemodynamics and peripheral organ function.[8]

Recently, clinical use of IABP has increased as a bridge to recovery or more intensive therapies such as durable ventricular assist devices (VADs) and heart transplantation.[9] Prior hemodynamic improvement is essential for successful outcomes. Although several studies showed that other enhanced mechanical circulatory supports are more effective in improving hemodynamics,[10,11] IABP still has the advantages of easy implant and explant, management simplicity, and cost effectiveness, with a very good safety profile.[12] These advantages may be even more evident when recently developed subclavian implantation approaches are employed.[13]

The next concern is which patients will achieve hemodynamic stabilization from IABP alone as a bridging strategy without any enhanced mechanical support. Another way to see the efficacy of IABP therapy would be assessing the degree of hemodynamic improvement, although such improvement may not always reach complete hemodynamic stabilization.

Accordingly, in this study, we investigated predictors of hemodynamic improvement or stabilization by IABP therapy to identify a therapeutic strategy to optimize selection for patients with advanced HF failing medical therapy.

Methods

Patient selection

In this retrospective study, clinical data were collected from all advanced HF patients who had received IABP implantation via subclavian approach to treat hemodynamic deterioration. Patients with acute coronary syndrome were excluded from this study. At our institution, patients with aortic dissection, aortic valve regurgitation, aortic aneurysm, or sepsis did not receive IABP implantation. All patients were followed until IABP removal. The study protocol was approved by the Institutional Review Board and written informed consent was obtained from all participants.

Variables evaluated

Baseline characteristics including demographic and laboratory data were collected immediately before IABP implantation. Serum levels of total bilirubin, creatinine, and lactic acid were obtained at the study endpoint to assess the improvement in peripheral circulation.

Echocardiographic data were obtained within 1 week before IABP implantation. Left ventricular ejection fraction was calculated by modified Simpson's method. The degree of valve regurgitation was graded as: 0, none; 1, trace; 2, mild; 3, moderate; or 4, severe.

Invasive hemodynamic studies were performed immediately before IABP implantation and at the study endpoint, which was defined as occurring after 2 weeks of IABP implantation or just before IABP termination within 2 weeks as a standard protocol. Cardiac index (CI) was calculated by the indirect Fick method.

Significant hemodynamic improvement was defined as >30% improvement following IABP implantation in all three variables (pulmonary capillary wedge pressure [PCWP], central venous pressure [CVP], and CI). *Hemodynamic stabilization* was defined as CVP <12 mm Hg, PCWP <18 mm Hg, and CI >2.0 L/min/m^2 or CI >2.2 L/min/m^2 with inotropes following IABP implantation.

Statistical analyses

Data are expressed as mean ± standard deviation, unless otherwise indicated. Continuous variables were compared using the unpaired t-test or Mann– Whitney U-test as appropriate, and categorical variables were compared using the Chi-square test or Fisher's exact test as appropriate. Changes in clinical variables during IABP therapy were analyzed using paired t-test. Logistic regression analyses were performed to investigate predictors for the endpoints. Dichotomous variables divided at the cutoff point calculated by using receiver operating characteristic curve analyses were adopted when continuous variables showed statistical significance. Significant dichotomous variables in univariate analyses were enrolled into multivariate analyses considering multicolinearity by using variance inflation factor. Improvements of hemodynamics and hemodynamic stabilization were assessed by McNemar test. All statistical analyses were performed using SPSS Statistics 22 (SPSS, Inc), and 2-tailed $P<.05$ was considered significant.

Results

Baseline characteristics

Ninety-one consecutive patients (55.4 ± 12.3 years; 78% males) were enrolled. Baseline characteristics are summarized in Table 1. One-half of patients (53.8%) had non-ischemic HF etiology, and 69.2% received inotrope infusions at the time of implant. Mean duration of IABP support was 25.1 ± 20.4 days, and 59 patients (64.8%) continued IABP support for ≥14 days.

Hemodynamic improvements in CVP, PCWP, and CI

PCWP decreased by 6.2 ± 9.8 mm Hg, CVP decreased by 2.4 ± 6.8 mm Hg, and CI increased by 0.436 ± 0.732 L/min/m^2 (*P*<.01 for all) (Figures 1A–1C). Mean percent changes in PCWP, CVP, and CI were −21.4 ± 35.6%, −2.6 ± 75.0% and 29.5 ± 49.3%, respectively. As a result, 17 patients (18.7%) satisfied the hemodynamic improvement endpoint of >30% improvement in all three hemodynamic variables.

Predictions of significant degree of hemodynamic improvement

Among all baseline variables, inotrope usage, higher CVP and PCWP, lower CI, and lower left ventricular (LV) cardiac power index were associated with significant hemodynamic improvement (Table 2). Particularly, CVP >16 mm Hg was a significant predictor for hemodynamic improvement at multivariate analysis (odds ratio [OR], 5.02). No echocardiographic parameters, including moderate to severe mitral regurgitation and right-ventricle associating index, were associated with hemodynamic improvement (*P*>.05 for all).

Patients with CVP >16 mm Hg (n = 23) had more severe biventricular failure with tachycardia despite inotrope support compared to those without (*P*<.05 for all) (Supplementary Table S1; all supplemental tables/figures available at www.invasivecardiology.com). Improvements in all three hemodynamic variables (PCWP, CVP, and CI) were significantly higher in patients with CVP >16 mm Hg compared to those without CVP >16 mm Hg (Figures 1D–1F) (*P*<.05 for all).

Hemodynamic stabilization

The percentages of patients satisfying the individual components of the composite endpoint for hemodynamic stabilization are summarized in Figure 2. At baseline, some patients satisfied individual components, but only 5 patients (5.5%) satisfied all three criteria. Following IABP support, 32 patients (35.2%) satisfied all three criteria.

Predictors for hemodynamic stabilization

Multivariate analysis showed that heart rate (HR) <92 beats/ min (OR, 4.08) and PCWP <25 mm Hg (OR, 4.66) were significant predictors for hemodynamic stabilization among all baseline variables (*P*<.05 for both) (Table 3). Compared to patients with HR ≥92 beats/min or PCWP ≥25 mm Hg, the patient group with such predictors had more females, lower rates of inotrope infusion, were less volume overloaded, and had more preserved LV contractility with lower HR as their baseline characteristics (Supplementary Table S2).

Thirty patients (33.0%) satisfied HR <92 beats/min and PCWP <25 mm Hg at baseline. The prevalence of hemodynamic stabilization increased significantly from 10.0% to 66.7% in this population (*P*<.05), whereas hemodynamic stabilization was achieved in only 19.7% of the remaining patients (*P*<.001 between the groups).

Changes in end-organ perfusion

Among all patients, serum total bilirubin level remained unchanged (*P*=.21) (Supplementary Figure S1A), serum creatinine level decreased (*P*<.01) (Supplementary Figure S1B), and lactic acid level increased during IABP support (*P*<.01) (Supplementary Figure S1C).

Among those with hemodynamic stabilization, serum total bilirubin decreased (from 0.95 ± 0.61 mg/dL to 0.69 ± 0.53 mg/dL; $P<.01$) and creatinine level also decreased (1.62 ± 1.12 mg/dL to 1.28 ± 0.79 mg/dL; $P=.048$), whereas lactic acid did not increase (from 1.76 ± 2.02 mmol/L to 1.30 ± 0.59 mmol/L; $P=.24$).

Endpoints and clinical outcomes

Eventually, 11 patients (12.1%) had hemodynamic recovery, 63 patients (69.2%) were bridged to ventricular assist device or transplant, 4 patients (4.4%) required enhanced mechanical circulatory supports, and 13 patients (8.6%) died (Supplementary Table S3).

All of the patients who had hemodynamic improvement reached the next therapy except for 1 patient who required enhanced mechanical circulatory support. All of those who had hemodynamic stabilization reached the next level of therapy except for 1 patient who died from sepsis. There was no significant difference in background variables between patients who required enhanced mechanical support or died and those who did not (data not shown).

Discussion

In this study, we investigated predictors for hemodynamic improvement and stabilization during IABP therapy in patients with advanced HF and hemodynamic deterioration and found the following: (1) The majority of the patients (81.3%) reached the next therapeutic phase. (2) Approximately 20% of patients achieved significant degree of hemodynamic improvement, and baseline higher CVP was a predictor of such an endpoint. (3) Approximately 35% of patients achieved hemodynamic stabilization. Lower baseline HR and PCWP were associated with such an endpoint. (4) No baseline demographic and echocardiographic characteristics were associated with both endpoints.

We assessed hemodynamic changes (hemodynamic improvement and stabilization) as endpoints instead of clinical outcomes such as the use of enhanced mechanical support.[9] Hemodynamic stabilization is an ideal goal for successful IABP therapy, whereas hemodynamic improvements, although not necessarily complete hemodynamic stabilization, may be another sign of successful IABP therapy.

Most of the patients with hemodynamic improvement achieved bridge to cardiac replacement therapy, but not myocardial recovery. We should understand that good response to IABP (ie, the degree of hemodynamic improvement is high) does not always reach the optimal clinical outcomes such as myocardial recovery. Nevertheless, we should remember the value of CVP >16 mm Hg, at which IABP can work best. Higher CVP/PCWP ratio and lower right ventricular (RV) stroke work index tended to be associated with better response to IABP. The mechanisms for which elevated CVP represents a responder are not clear, but may suggest that IABP is working better in high-volume status rather than low-flow state. In such situations, patients are too sick, and may often have biventricular failure despite inotrope support, as we showed.

In contrast, patients with hemodynamic stabilization achieved myocardial recovery more frequently than those with hemodynamic improvement. Stabilized hemodynamics and end-

organ function before the surgery may result in successful cardiac replacement therapies accompanied by better clinical outcomes.[14,15]

Lower baseline HR and PCWP were strong predictors of hemodynamic stabilization; these patients had less severe HF, and thus were less sick. It is easy to understand that less-sick patients have a better chance to achieve hemodynamic stabilization, because their hemodynamics are already near the goal before IABP. Both HR and PCWP may be good indicators to understand the severity of HF.[16] It is reasonable that HF patients who are too sick and over this threshold have less chance to normalize by IABP alone, even though these sick patients may experience sufficient hemodynamic improvement.

Recently, Sintek et al demonstrated that higher RV and LV cardiac power indices and higher systolic pulmonary artery pressure may help predict patients who will be clinically stabilized during IABP support.[9] Those findings are easy to explain because IABP is an augmentation device, and better power indices represent a less sick patient population. However, our definition of a responder represents changes or stabilization in hemodynamics and does not take into account whether the patients did not deteriorate if the IABP successfully did what it is supposed to do. In their study, hemodynamic changes and final hemodynamic levels were comparable between the clinically stabilized group and the clinically deteriorated group.

In the previous report, IABP reduced 30-day mortality in patients with cardiogenic shock following myocardial infarction and mitral regurgitation.[17] Although the clinical situation is different, patients with moderate to severe mitral regurgitation tended to have hemodynamic improvement in our study as well (P=.09). We believe that patients with advanced mitral regurgitation may benefit from IABP; however, larger studies are warranted.

Krishnamoorthy et al reported that patients with biventricular failure more frequently required early escalation of mechanical support within 3 days of IABP implantation than those with left-sided HF, although there were no significant differences with regard to 30-day outcomes.[18] Our study design focused on the hemodynamics and not on the need for escalating care; however, we identified that patients with biventricular failure had more significant degree of hemodynamic improvement (which does not mean that this improvement was enough for hemodynamic stabilization). In line with this finding, Ntalianis et al reported that prolonged IABP support in patients with advanced HF and RV dysfunction induced significant improvement in RV function.[8]

Currently, we can use various temporary mechanical circulatory support devices, and should select optimal devices that improve hemodynamics and maintain patient comfort.[12] The identification of predictors will help clinicians select IABP alone or more aggressive mechanical therapies at the time of hemodynamic measurement before the initiation of any treatment. The ideal goal is long-term prognosis after the next therapeutic phase following IABP support. Long-term implications of hemodynamic improvement and hemodynamic stabilization should be clarified in the next study.

All patients received IABP support via subclavian approach, which helps them stay mobile, improves their comfort, and helps them participate in active physical therapy if necessary.[13]

The portable NuPulseCV intravascular assist IABP system may be available shortly.[19] The prediction of hemodynamic stabilization would be particularly useful for this kind of ambulatory therapy, in which more safety is required. Also, such predictors may be useful to select good candidates for the concomitant aggressive cardiac rehabilitation during IABP support. IABP therapy for >2 weeks may be possible using this novel system.

Study limitations

This is a retrospective, single-center experience with a relatively small number of patients. The findings need to be confirmed in a future multicenter, large-scale study. We assessed only patients with advanced HF, and our results should not simply be adopted in patients with acute *de novo* HF, acute coronary syndrome, or pure right-sided HF. During the study periods, we did not take into account medication changes during IABP therapy.

Conclusion

IABP provides significant improvement in hemodynamics in 20% of chronic HF patients. Elevated CVP is a predictor for significant hemodynamic response to IABP. Furthermore, 35% of the patients achieved hemodynamic stabilization; those patients tend to be less sick (lower HR and lower PCWP). Utilizing preimplantation hemodynamics for device selection may increase the success rate of treating decompensated HF patients.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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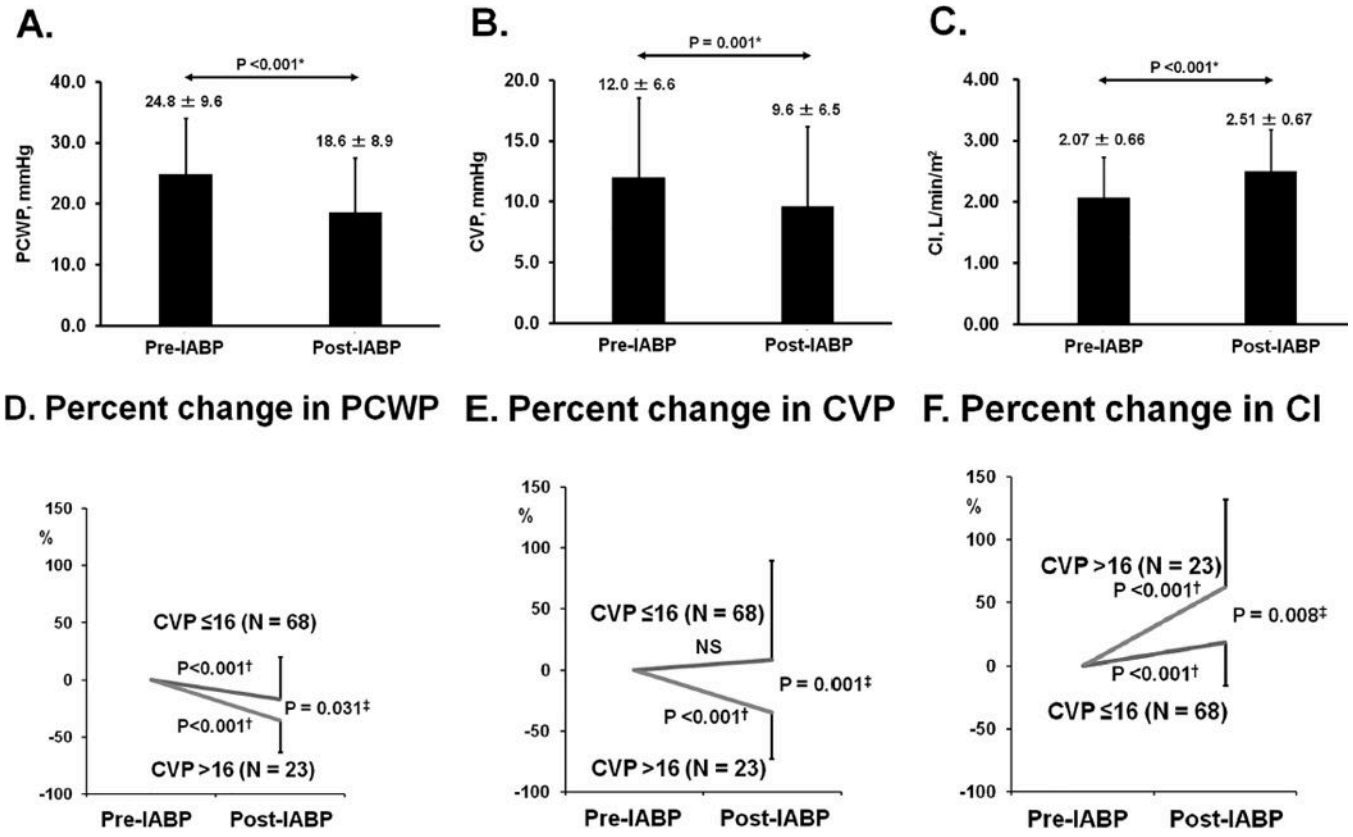

FIGURE 1.

Changes in hemodynamic variables pre and post intraaortic balloon pump (IABP): (A) pulmonary capillary wedge pressure (PCWP); (B) central venous pressure (CVP); and (C) cardiac index (CI). Percentage changes between hemodynamic variables pre and post IABP: (D) PCWP; (E) CVP; and (F) CI stratified the baseline CVP level. $^{*}P<.05$ by paired t-test; $^{\dagger}P<.05$ by paired t-test between pre and post variables; $^{\ddagger}P<.05$ between the groups by unpaired t-test or Mann–Whitney test as appropriate.

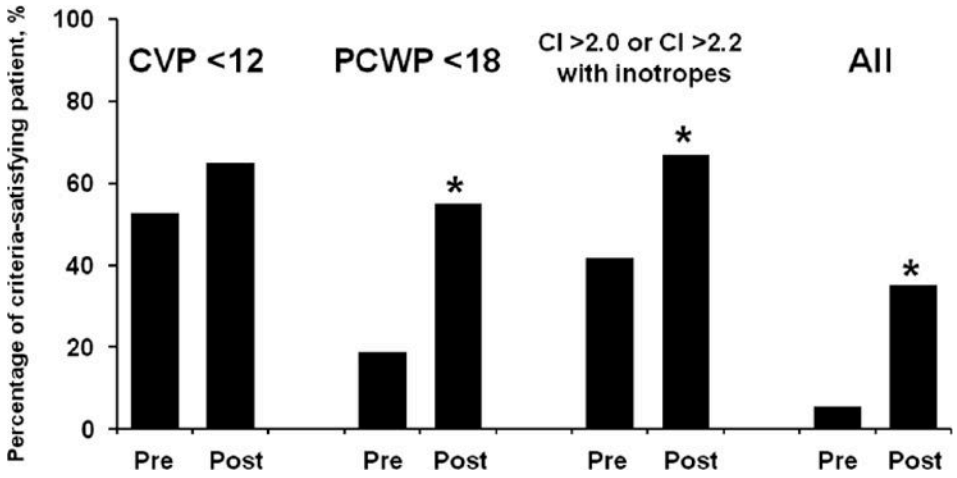

FIGURE 2.

Percentage of criteria-satisfying patients pre and post intraaortic balloon pump stratified by each criterion. $^{*}P<.05$ by McNemar test. CVP = central venous pressure; PCWP = pulmonary capillary wedge pressure; CI = cardiac index.

Table 1

Baseline characteristics.

	n = 91
Demographics	
Age (years)	55.4 ± 12.3
Male gender	71 (78.0%)
Non-ischemic etiology	49 (53.8%)
Body surface area (m^2)	1.94 ± 0.25
Inotropes infusion	63 (69.2%)
Echocardiography	
Left ventricular diastolic diameter (cm)	6.9 ± 1.2
Left ventricular ejection fraction (%)	21.4 ± 9.3
Mitral regurgitation (grade)	2.4 ± 1.2
Mitral regurgitation with moderate to severe grade	47 (51.6%)
Tricuspid regurgitation (grade)	2.0 ± 1.2
Hemodynamics	
Mean arterial pressure (mm Hg)	83.5 ± 12.4
Heart rate (beats/min)	92.5 ± 15.1
Central venous pressure (mm Hg)	12.0 ± 6.6
Mean pulmonary artery pressure (mm Hg)	41.7 ± 11.6
Pulmonary capillary wedge pressure (mm Hg)	24.8 ± 9.2
Cardiac index ($L/min/m^2$)	2.07 ± 0.66
Left ventricular cardiac power index (W/m^2)	0.37 ± 0.12
Right ventricular cardiac power index (W/m^2)	0.19 ± 0.07
Central venous pressure/pulmonary capillary wedge pressure ratio	0.48 ± 0.20
Right ventricular stroke work index (g/m^2)	9.30 ± 4.59
Pulmonary artery pulsatility index	2.84 ± 2.63
Laboratory values	
Serum creatinine (mg/dL)	1.81 ± 1.78
Serum total bilirubin (mg/dL)	1.19 ± 1.10

Data provided as mean ± standard deviation or number (percentage).

Table 2

Logistic regression analyses for significant hemodynamic improvement among baseline variables (only significant variables are shown).

	Univariate Analysis		Multivariate Analysis		VIF
	***P*-Value**	**Odds Ratio (95% CI)**	***P*-Value**	**Odds Ratio (95% CI)**	
Continuous variables					
Central venous pressure (mm Hg)	<.01 *	1.14 (1.05–1.24)			
Pulmonary capillary wedge pressure (mm Hg)	.03 *	1.07 (1.01–1.13)			
Cardiac index (L/min/m^2)	<.01 *	0.19 (0.057–0.65)			
Left ventricular cardiac power index (W/m^2)	.01 *	0.001 (0.001–0.12)			
Dichotomous variables					
Inotrope infusion	.04 *	9.19 (1.15–73.2)	.29	3.37 (0.35–32.4)	1.37
Central venous pressure >16 mm Hg	<.01 *	6.70 (2.15–20.9)	.02 *	5.02 (1.27–19.9)	1.20
Pulmonary capillary wedge pressure >19 mm Hg	.07	6.77 (0.85–54.2)			
Cardiac index <1.43 L/min/m^2	<.01 *	6.70 (1.94–23.2)	.10	7.87 (0.65–94.8)	2.07
Left ventricular cardiac power index <0.29 W/m^2	.02 *	4.19 (1.24–14.2)	.65	0.58 (0.052–6.31)	2.05

VIF = variance inflation factor; CI = confidence interval.

* $P<.05$ by logistic regression analyses.

J Invasive Cardiol. Author manuscript; available in PMC 2018 March 22.

Table 3

Logistic regression analyses for hemodynamic stabilization among baseline variables (only significant variables are shown).

	Univariate Analysis		Multivariate Analysis		VIF
	***P*-Value**	**Odds Ratio (95% CI)**	***P*-Value**	**Odds Ratio (95% CI)**	
Continuous variables					
Heart rate (beats/min)	.02*	0.96 (0.93–0.99)			
Central venous pressure (mm Hg)	<.01*	0.87 (0.77–0.95)			
Mean pulmonary artery pressure (mm Hg)	<.01*	0.92 (0.88–0.97)			
Pulmonary capillary wedge pressure (mm Hg)	<.001*	0.87 (0.81–0.94)			
Cardiac index (L/min/m^2)	<.01*	4.56 (1.78–11.7)			
Pulmonary artery pulsatility index	.03*	1.23 (1.02–1.48)			
Dichotomous variables					
Heart rate <92 beats/min	<.02*	4.38 (1.69–11.4)	.02*	4.08 (1.25–13.3)	1.23
CVP <9 mm Hg	<.001*	7.27 (2.76–19.2)	.18	3.27 (0.59–18.2)	2.79
Mean pulmonary artery pressure <37 mm Hg	<.001*	6.38 (2.44–16.7)	.26	2.09 (0.59–7.47)	1.81
Pulmonary capillary wedge pressure <25 mm Hg	<.001*	10.5 (3.52–31.5)	.02*	4.66 (1.29–16.9)	2.71
Cardiac index >1.85 L/min/m^2	<.01*	4.48 (1.61–12.5)	.09	2.98 (0.83–10.7)	1.18
Pulmonary artery pulsatility index >2.45	.03*	2.71 (1.12–6.57)	.64	0.677 (0.13–3.52)	1.87

VIF = variance inflation factor; CI = confidence interval.

*P<.05 by logistic regression analyses.