Comparison of the Hemodynamic Response to Intra-Aortic Balloon Counterpulsation in Patients With Cardiogenic Shock Resulting from Acute Myocardial Infarction Versus Acute Decompensated Heart Failure

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The intra-aortic balloon pump (IABP) neither benefits nor harms patients with acute myocardial infarction (AMI) with cardiogenic shock (CS) but may stabilize those with chronic heart failure who decompensate into CS. We sought to compare its hemodynamic effects in these 2 populations. We performed a retrospective analysis of the hemodynamic effects of IABP for AMI or acute decompensated heart failure (ADHF) patients with hemodynamic evidence of CS. The primary outcome was cardiac output (CO) change following insertion. In total, 205 patients were treated for CS resulting from AMI (73; 35.6%) or ADHF (132; 64.4%). At baseline, both cohorts had significant hemodynamic compromise with mean arterial pressure 75.6 ± 12.3 mm Hg, CO 3.02 ± 0.84 L/min, and cardiac power index 0.26 ± 0.06 W/m^2; these parameters were nearly identical between groups though ADHF-CS patients had a higher pre-IABP mean pulmonary artery (PA) pressure than AMI-CS patients. After IABP insertion, ADHF-CS patients had moderate CO augmentation whereas AMI-CS experienced almost no improvement (0.58 ± 0.79 L/min vs 0.12 ± 1.00 L/min; p = 0.0009). Intracardiac filling pressures were reduced by similar amounts in both cohorts. Systemic vascular resistance was reduced in patients with ADHF-CS but not in those with AMI-CS. In conclusion, following IABP insertion, ADHF-CS patients experience roughly a 5-fold greater CO augmentation compared with AMI-CS patients. Pre-IABP PA pressure differences and differential systemic vascular resistance reduction may explain these results and shed light on recent evidence supporting IABP use in ADHF-CS and curbing it in AMI-CS. **(Am J Cardiol 2019;124:1947−1953)**

Despite advances in medical- and device-based therapies, the prognosis of cardiogenic shock (CS) remains poor.[1] Temporary mechanical support devices (MCSDs) are often utilized in CS refractory to medical therapy to stabilize patients sufficiently, then wean from the device or bridge to heart replacement therapy (HRT) such as a durable left ventricular assist device (LVAD) or heart transplantation (HT). Commonly used percutaneous options include the intra-aortic balloon pump (IABP), percutaneous ventricular assist device (pVAD), and extracorporeal membrane oxygenation (ECMO).[2] Of these, the IABP is used most commonly due to its widespread availability, ease of insertion, and low complication rate.[3] Despite decades of IABP experience, its optimal use is poorly defined. It has been shown to be of neither benefit nor harm for CS following AMI. However, there is new interest in its use to stabilize patients with acutely decompensated heart failure (ADHF) with CS.[4,5] We sought to compare the hemodynamic response to IABP insertion in patients with CS from either ADHF or AMI to determine whether this response might differ between different etiologies of CS.

Methods

We retrospectively reviewed medical records of all patients in the cardiac care unit who underwent IABP implantation at our institution from January 2011 to April 2016. We identified patients ≥18 years of age with AMI or ADHF and hemodynamic evidence of CS defined as pre-IABP cardiac index (CI) <2.2 L/min/m^2 and either systolic blood pressure (SBP) <90 mm Hg or need for vasoactive medications to achieve this blood pressure. We restricted our population to those with hemodynamic evidence of CS as the study goal was to understand the hemodynamic effects of the IABP. ADHF was defined as an acute presentation of a patient with left ventricular ejection fraction (LVEF) ≤40% for ≥6 months. AMI patients included those

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with ST-elevation myocardial infarction and non-ST elevation myocardial infarction. Patients were excluded from analysis if (1) IABP placement occurred after cardiac surgery; (2) support with another MCSD had occurred before IABP implantation (e.g., ECMO); or (3) preimplant hemodynamics were not obtained. The study was approved by the Columbia University Institutional Review Board.

Demographic data for the ADHF-CS and AMI-CS cohorts were collected, including co-morbidities and echocardiographic parameters. For the AMI-CS cohort, angiographic data, culprit vessel, and type of revascularization (e.g., coronary artery bypass graft surgery [CABG] vs percutaneous coronary intervention [PCI]) were also identified. Hemodynamic data included pulmonary artery (PA) catheter measurements, including cardiac output (CO) and CI by Fick method, cardiac power output/index (CPO/CPI), PA pulsatility index (PAPi). Change in CO, change in CI, and percent change in CO were calculated based on the pre- and post-IABP implantation hemodynamic differences.

The primary outcome was CO change as a marker of hemodynamic response to IABP implantation. Secondary outcomes included other hemodynamic parameters including CPO and CPI, in-hospital mortality, and the need for escalation to another MCSD. Continuous variables are presented as mean ± standard deviation or median with interquartile range and were compared using the Student's *t* test or Wilcoxon's rank sum test, as appropriate. For outcomes of interest, continuous variables were compared using ANCOVA to control for important covariates. Categorical variables are presented as percentages and were compared using chi-square tests. All analyses were performed using STATA version 15.0 (StataCorp LP, College Station, TX).

Results

Figure 1 details IABP use at our institution during the study period. Overall, 701 patients underwent IABP implantation from January 2011 to April 2016. Of these, 396 patients presented with AMI and 202 presented with ADHF. Seventy-three AMI patients and 132 ADHF patients met our predefined inclusion criteria with hemodynamic evidence of pre-IABP CS. Of those with AMI, 323 patients were excluded for the following reasons: incomplete hemodynamic data (n = 225) and preimplant hemodynamics not consistent with CS (n = 98). Of 202 ADHF patients, 70 were excluded for the following reasons: previous HT (n = 13), lack of complete preimplant hemodynamics (n = 32), and hemodynamics not consistent with CS (n = 25). Our annual institutional utilization of IABP for these 2 etiologies of CS is represented in Figure 2. IABP use in ADHF-CS rose steadily during the study period whereas it declined for AMI-CS.

The study cohort's baseline demographics and echocardiographic characteristics are presented in Table 1. There was a significant difference in age, gender, body surface area (BSA), diabetes mellitus (DM) prevalence, left ventricular ejection fraction (LVEF), and left ventricular end-diastolic diameter (LVEDd) between the ADHF-CS and AMI-CS groups. ADHF-CS patients had a higher serum creatinine at baseline but lower serum lactate than AMI-CS patients.

Procedural characteristics for patients who underwent IABP implantation for AMI-CS are presented in Table 2. The left anterior descending (LAD) or left main (LM) coronary artery was the culprit vessel in 46.6% of the cases. The burden of coronary artery disease was high with an average of 2.2 ± 0.8 coronary vessels narrowed (defined as >50% stenosis of a major epicardial vessel). Twenty-two (30.1%) AMI patients had IABP implanted after revascularization as opposed to before it. For 7 patients, revascularization could not be performed (either unsuccessful, deferred, or the patient expired before revascularization).

Baseline hemodynamic characteristics are displayed in Table 3. The overall cohort manifested significant hemodynamic compromise with a mean arterial blood pressure (MAP) of 75.6 ± 12.3 mm Hg on 1.6 ± 1.0 vasopressors/inotropes

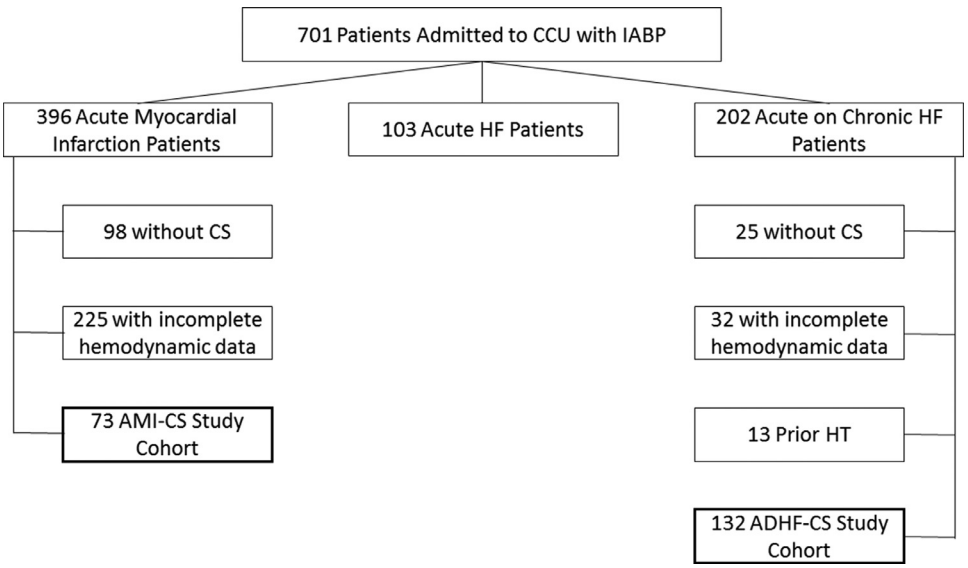

Figure 1. Institutional use of IABP. The study cohort was derived from an analysis of all IABP use in the CCU from January, 2011 to April, 2016. Patients with AMI or ADHF and hemodynamic evidence of CS before IABP insertion were included. ADHF, acute decompensated heart failure; AMI, acute myocardial infarction; CCU, cardiac intensive care unit; CS, cardiogenic shock; HF, heart failure; HT, heart transplant.

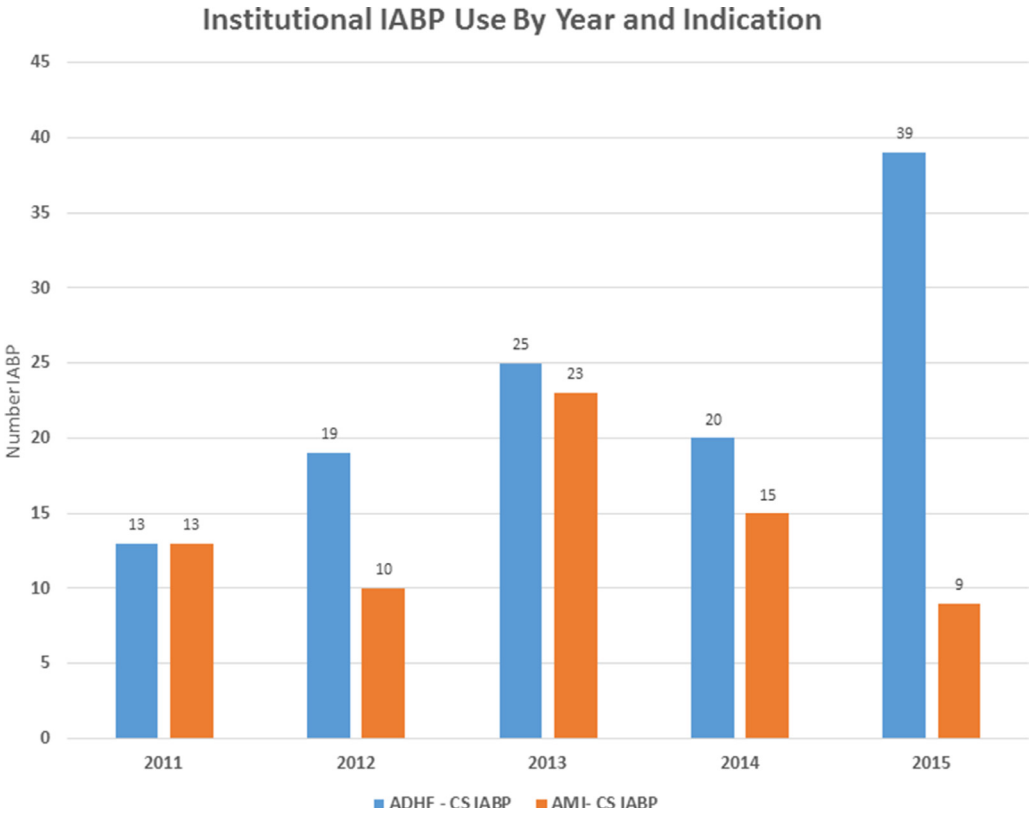

Figure 2. Institutional IABP use by year and indication. The use of IABP for AMI decreased during the study period while its use for ADHF increased. ADHF, acute decompensated heart failure; AMI, acute myocardial infarction; CS, cardiogenic shock.

Table 1
Baseline demographics for patients with IABP placed for cardiogenic shock

	All (n = 205)	ADHF-CS (n = 132)	AMI-CS (n = 73)	p Value
Age (y)	65.0 ± 13.8	61.2 ± 13.0	71.8 ± 12.7	<0.0001
Men	148 (72.2%)	111 (84.1%)	37 (50.7%)	<0.001
Body surface area (m^2)	1.90 ± 0.24	1.93 ± 0.25	1.85 ± 0.22	0.02
Diabetes mellitus	82 (40.0%)	44 (33.3%)	38 (52.1%)	0.009
Left ventricular ejection fraction (percent)	22.2 ± 11.7	18.0 ± 8.9	30.2 ± 12.2	<0.0001
Left ventricular end-diastolic diameter (cm)	6.2 ± 1.5	7.0 ± 1.1	4.8 ± 0.9	<0.0001
Serum creatinine (mg/dL)	1.83 ± 1.09	1.97 ± 1.06	1.59 ± 1.11	0.02
Serum lactate (mg/dL)	3.65 ± 3.58	2.54 ± 2.50	4.92 ± 4.21	0.003

ADHF-CS, acute decompensated heart failure - cardiogenic shock; AMI-CS, acute myocardial infarction - cardiogenic shock.

with a CI of 1.58 ± 0.39 L/min/m^2 and a CPI of 0.26 ± 0.07 W/m^2. The MAP was nearly identical between those with AMI and ADHF though the latter had a lower pulse pressure. The CO and CPI were also nearly identical for the 2 cohorts. The baseline stroke volume (SV) was 33.9 ± 11.9 mL in ADHF-CS patients and 35.4 ± 12.4 mL in AMI-CS patients (p = 0.40). Systemic vascular resistance (SVR) was similar between the 2 cohorts (1,680.2 ± 541.4 dyn·s·cm^{-5} for ADHF-CS patients vs 1,745.1 ± 821.8 dyn·s·cm^{-5} for AMI-CS patients, p = 0.52). However, pulmonary artery pressures and PAPi were higher in the ADHF cohort.

Figure 3 displays the CO change with IABP for each patient and the overall hemodynamic changes observed with IABP insertion are displayed in Table 4. The median duration of IABP support between pre- and postinsertion measurements was 5.0 hours (interquartile range [IQR]: 3.5 to 9.0). The mean number of vasoactive infusions administered during postinsertion measurements was 1.5 ± 1.1; 27 patients (14.2%) had an increase in the number of inotropic or vasopressor medications between the pre- and postimplant measurements whereas 115 (60.5%) had no change and 48 (25.3%) had a reduction in these medications. No patients received vasodilator therapy during the periimplantation period.

The mean CO change for the entire cohort was 0.44 ± 0.88 L/min. However, there was a significant difference in the mean augmentation between the ADHF-CS and AMI-CS cohorts with the former experiencing almost a 5-fold greater CO augmentation compared with the latter (0.58 ± 0.79 L/min vs 0.12 ± 1.00 L/min, p = 0.0009). This amounted to only a 10% CO increase for AMI-CS patients whereas those with ADHF-CS experienced an increase by almost a quarter of their

Table 2
Procedural characteristics for acute myocardial infarction patients

Variable	AMI-CS
Access	
Femoral	73 (100%)
Axillary	0
Acute coronary syndrome type	
ST elevation myocardial infarction	40 (54.8%)
Non-ST elevation myocardial infarction	33 (45.2%)
Timing of IABP	
Prepercutaneous coronary intervention	51 (69.9%)
Postpercutaneous coronary intervention	22 (30.1%)
Culprit coronary artery	
Right	9 (12.3%)
Left circumflex	5 (6.9%)
Left main or left anterior descending	34 (46.6%)
Multiple coronary arteries	25 (34.3%)
Number coronary arteries narrowed	2.2 ± 0.8
Revascularization	
Percutaneous coronary intervention	59 (80.8%)
Coronary artery bypass graft surgery	7 (9.6%)
Revascularization not performed	7 (9.6%)

AMI, acute myocardial infarction; CS, cardiogenic shock.

baseline CO. Among those in the ADHF cohort, there was a trend toward a greater CO increase with IABP insertion in patients with a nonischemic dilated cardiomyopathy as opposed to an ischemic dilated cardiomyopathy (0.68 ± 0.72 L/min vs 0.40 ± 0.89 L/min, p = 0.052). We examined univariable predictors of CO change with IABP insertion and included these in an ANCOVA model to control for potential baseline differences in the cohorts. After controlling for age, gender, BSA, DM prevalence, vasopressor/inotropic medications, baseline CO, mean PA pressure (mPAP), LVEDd, and SV, the underlying etiology of CS (i.e., AMI vs ADHF) remained a significant predictor of IABP hemodynamic response (p = 0.03).

In addition to CO augmentation, we examined PA pressure reduction with IABP insertion and found similar mPAP reduction in the cohorts with IABP therapy. There was negligible change in MAP overall and no difference in MAP change between the cohorts. Lastly, the SVR difference between the pre- and postimplant hemodynamic evaluation was −253.1 ± 493.0 dyn·s·cm^{-5} in ADHF-CS patients and 21.3 ± 843.0 dyn·s·cm^{-5} for AMI-CS patients (p = 0.01).

Figure 4 displays the clinical outcomes of study patients. The median duration of IABP support was 3.0 days (IQR: 2.0 to 5.0). In the AMI-CS cohort, 53 patients (72.6%) survived to discharge including 1 who required durable LVAD implantation. Of 73 patients with AMI-CS, 47 patients (64.4%) survived to discharge with only IABP for mechanical circulatory support, whereas 14 (19.2%) died without escalation to another MSCD. Twelve patients (16.4%) had another MCSD implanted after IABP. Of these, 5 received a pVAD and 7 received VA-ECMO and of those initially receiving pVAD, 2 patients also had VA-ECMO implanted due to persistent CS. Of those requiring escalation to either of these devices, 6 (50.0%) died. Lastly, the median time on IABP support was 2.0 days (IQR: 2.0 to 4.0).

In contrast to those with AMI-CS, the vast majority of ADHF-CS patients underwent HRT during the index admission. Although a similar number of patients (10, 7.6%) required escalation to another more potent short-term circulatory support device, 87 patients ultimately underwent HRT initiation (11 HT and 76 durable LVAD). Overall, 103 (78.0%) ADHF-CS survived to discharge with or without HRT. The median time on IABP support for those with ADHF-CS was 4.0 days (IQR: 2.0 to 6.0).

Discussion

In this study, we performed a comprehensive examination of the hemodynamic response to IABP implantation in patients presenting with AMI-CS and ADHF-CS. Our principal finding is that AMI-CS patients had minimal CO augmentation in response to IABP insertion, whereas those with ADHF-CS had moderate CO augmentation which was roughly a 5-fold increase compared with that seen with AMI-CS. To our knowledge, this is the first comparison of the hemodynamic response to aortic counterpulsation between 2 commonly supported patient phenotypes.

IABPs have been implanted for CS for >40 years. The earliest studies contained a heterogeneous population and demonstrated improvements in CO of roughly 0.5 L/min in

Table 3
Baseline hemodynamic data for patients with IABP placed for cardiogenic shock

	All (n = 205)	ADHF-CS (n = 132)	AMI-CS (n = 73)	p Value
Systolic blood pressure (mm Hg)	101.8 ± 18.2	98.4 ± 14.6	108.0 ± 22.2	0.0003
Diastolic blood pressure (mm Hg)	62.6 ± 12.7	64.2 ± 10.9	59.7 ± 15.2	0.02
Mean arterial pressure (mm Hg)	75.6 ± 12.3	75.6 ± 10.6	75.7 ± 14.8	0.97
Cardiac output (L/min)	3.02 ± 0.84	3.01 ± 0.78	3.02 ± 0.93	0.91
Cardiac index (L/min/m^2)	1.58 ± 0.39	1.56 ± 0.36	1.62 ± 0.44	0.33
Cardiac power output (W)	0.50 ± 0.16	0.50 ± 0.14	0.51 ± 0.18	0.87
Cardiac power index (W/m^2)	0.26 ± 0.06	0.26 ± 0.06	0.27 ± 0.09	0.32
Central venous pressure (mm Hg)	14.3 ± 6.7	14.6 ± 7.2	13.6 ± 5.6	0.32
Pulmonary artery systolic pressure (mm Hg)	52.4 ± 14.5	56.6 ± 14.0	44.9 ± 12.3	<0.0001
Pulmonary artery diastolic pressure (mm Hg)	26.6 ± 8.1	28.2 ± 8.1	23.7 ± 7.3	0.0001
Mean pulmonary artery pressure (mm Hg)	35.3 ± 9.6	37.9 ± 9.3	30.7 ± 8.2	<0.0001
Pulmonary artery pulsatility index	2.59 ± 2.91	2.91 ± 3.35	2.00 ± 1.69	0.04
Systemic vascular resistance (dyn·s·cm^{-5})	1,702.3 ± 649.0	1,680.2 ± 541.4	1,745.1 ± 821.8	0.52
Vasoactive agents (number)	1.6 ± 1.0	1.7 ± 1.0	1.4 ± 0.8	0.048

ADHF, acute decompensated heart failure; AMI, acute myocardial infarction; CS, cardiogenic shock.

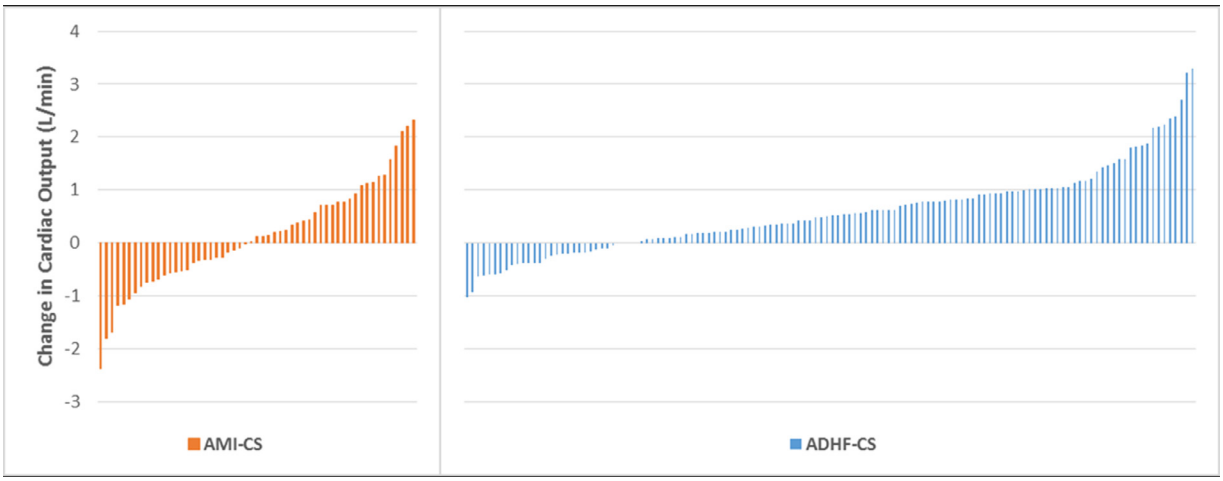

Figure 3. Cardiac output change following IABP insertion. The cardiac output change is displayed for each patient with AMI or ADHF. AMI, acute myocardial infarction; ADHF, acute decompensated heart failure; CS, cardiogenic shock.

Table 4
Hemodynamic changes observed with IABP insertion

	All (n = 205)	ADHF-CS (n = 132)	AMI-CS (n = 73)	p Value
Mean arterial pressure (mm Hg)	−0.5 ± 15.0	−1.4 ± 13.9	1.3 ± 17.1	0.26
Change in cardiac output (L/min)	0.44 ± 0.88	0.58 ± 0.79	0.12 ± 1.00	0.0009
Change in cardiac index (L/min/m^2)	0.24 ± 0.46	0.30 ± 0.42	0.08 ± 0.51	0.003
Cardiac output percent change (%)	20.0 ± 36.1	23.9 ± 35.2	10.1 ± 36.6	0.02
Cardiac power output (W)	0.07 ± 0.18	0.09 ± 0.17	0.03 ± 0.20	0.06
Cardiac power index (W/m^2)	0.04 ± 0.10	0.05 ± 0.0.9	0.02 ± 0.11	0.09
Central venous pressure (mm Hg)	−2.1 ± 5.6	−2.0 ± 5.2	−2.5 ± 6.6	0.59
Mean pulmonary artery pressure (mm Hg)	−5.2 ± 7.5	−5.3 ± 7.4	−5.0 ± 7.6	0.78
Systemic vascular resistance (dyn•s•cm^{-5})	−173.3 ± 625.5	−253.1 ± 493.0	21.3 ± 843.0	0.01
Vasoactive agents (number)	−0.1 ± 0.9	−0.1 ± 0.8	−0.2 ± 1.0	0.30

ADHF, acute decompensated heart failure; AMI, acute myocardial infarction; CS, cardiogenic shock.

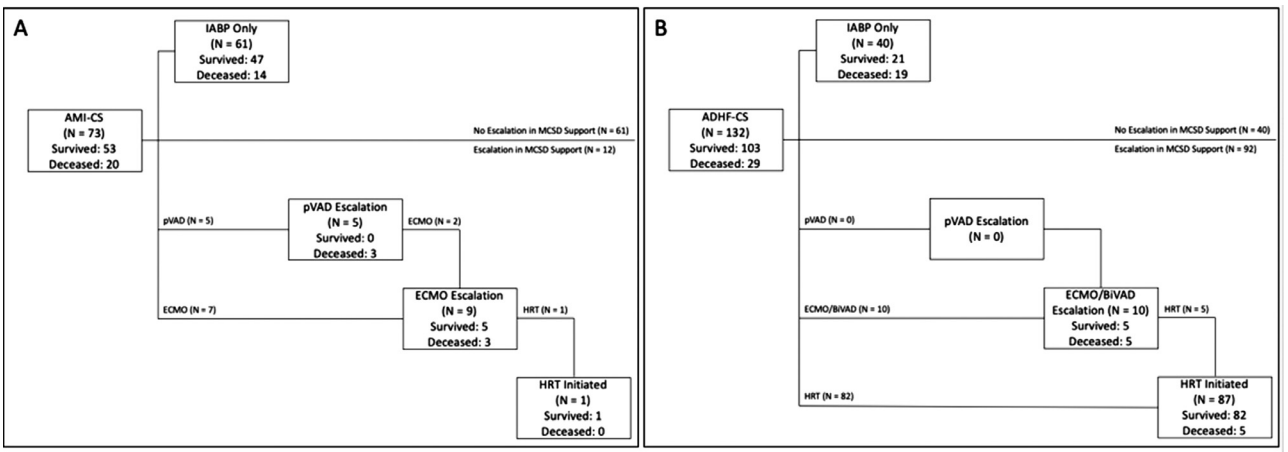

Figure 4. Patient outcomes following IABP insertion for cardiogenic shock. Patient outcomes are displayed following (A) AMI with CS and (B) ADHF with CS according to the MCSDs used. AMI, acute myocardial infarction; BiVAD, biventricular assist device (short-term, surgically implanted); CS, cardiogenic shock; ECMO, extracorporeal membrane oxygenation; HRT, heart replacement therapy; MCSD, mechanical circulatory support device; pVAD, percutaneous ventricular assist device.

CS patients.[6,7] We observed a similar CO change in our cohort but noted a large discrepancy between patients with different CS etiologies. Recent evidence has highlighted the importance of CS etiology[8,9]; the CardShock registry demonstrated a worse prognosis in AMI-CS compared with other CS etiologies. Thus, it should not be surprising that the response to IABP may differ by etiology as well. Although the hemodynamics are similar in each cohort, the differential IABP response suggests that these phenotypes are quite different.

Our findings are consistent with previous studies demonstrating negligible CO augmentation with IABP in AMI-CS.[10–12] Together with these data, ours suggests that in AMI-CS, IABP does little to improve hemodynamics. In this way, our study sheds light on the previous studies that demonstrated lack of clinical efficacy of IABP in AMI-CS. Notably the IABP-SHOCK II trial demonstrated no reduction in 30-day mortality with IABP for AMI-CS.[13] Hemodynamic measurements were not required for trial inclusion but our findings may explain why no benefit was observed. This finding is especially noteworthy in light of data highlighting the importance of CO and its derivatives in predicting outcomes in CS.[14,15]

Despite the lack of benefit for AMI-CS patients, there has been growing interest in IABP use for ADHF-CS patients. This has been fueled by the observation that many such patients can be stabilized by IABP with favorable clinical outcomes, particularly when bridged to HRT.[4,5] Although the mean CO augmentation in ADHF-CS patients was slightly >0.5 L/min, this may be sufficient improvement to stabilize a chronic heart failure patient with a relatively low CO at baseline. We have previously demonstrated that ADHF-CS patients were stabilized with IABP at low complication rates and characterized robust hemodynamic response in this cohort.[4] Sintek et al showed a >50% stabilization rate with IABP in patients with chronic heart failure bridged to LVAD.[5] Furthermore, new counterpulsation devices implanted in the subclavian artery have led to successful (and even long term) bridging to HRT, recovery, and improvement in biventricular function.[16–19]

Although our data suggest that different CS etiologies may have significantly different responses to IABP, the reason for this is not obvious. One explanation historically proposed is the need for intrinsic pulsatility for effective IABP support. However, the SV was almost identical between the 2 cohorts despite a difference in arterial pulse pressure. SVR was also comparable between the 2 populations, though ADHF patients experienced a reduction in SVR whereas AMI patients did not. The differential in response may be related to differential PA pressures and SVR reduction. Not surprisingly, those with ADHF-CS had significantly higher mPAP than those with AMI-CS. By reducing afterload, the IABP may allow for more forward flow in the setting of higher filling pressures. This would fit with our previous observation that the strongest predictor of IABP "super-response" (i.e., robust CO augmentation) was an elevated mPAP.[4] Another possible explanation is that those with greater right ventricular (RV) contractility, manifested by a higher PAPi which we observed in the ADHF-CS cohort at baseline, are primed to have greater response to IABP which does not directly support the RV. Thus, PAPi might be a good marker for patients with an expected favorable IABP hemodynamic response, as we have previously demonstrated.[4]

The outcomes of our AMI-CS patient cohort reflect that of a population with severe hemodynamic compromise including a CPI comparable to that observed in the SHOCK trial and registry and a significantly elevated serum lactate.[14] Importantly, a subset of patients underwent escalation to another MCSD and had a worse prognosis (as expected) than the overall AMI-CS cohort. Although ADHF-CS patients appear to derive greater hemodynamic support from the IABP than those with AMI-CS, the IABP alone is unlikely to stabilize ADHF-CS patients with more advanced forms of CS. The most hemodynamically unstable ADHF-CS patients typically require more powerful MCSDs.[20] However, the utilization of IABP for CS at our institution has shifted in response to the growing literature supporting use of IABP for ADHF-CS and the data supporting lack of benefit for AMI-CS patients. Our data provide hemodynamic evidence supporting these changes which mirror national trends with a decline in IABP use for AMI-CS and a rise in use of other MCSDs for the same indication.[21]

Our study has several significant limitations. First, our data represent a single-center experience and are retrospective in nature. Importantly, there were patients treated with IABP during the study lacking preimplantation hemodynamic data who were excluded from analysis. Though this resulted in a selected patient population, our goal was to understand hemodynamic effects of this device in CS and therefore, we restricted our inclusion criteria to those with hemodynamic evidence of this state. Decisions regarding escalation to other MCSDs reflect physician practice and were not protocolized for a portion of the study period. Detailed information regarding which vasopressors and inotropes were used for the entire cohort were not available to further understand the differential effects of the device on the SVR. Lastly, the timing of pre- and postimplantation hemodynamics were not uniform, nor were vasoactive medications held constant in all cases, limiting our ability to attribute all hemodynamic changes to the IABP. However, the majority of patients had either no change or a reduction in the number of vasoactive medications administered between the hemodynamic time points.

In conclusion, patients presenting with AMI-CS have minimal hemodynamic improvement with IABP insertion whereas those with ADHF-CS have a more robust improvement with this intervention. The reasons for this discrepancy remain unclear but may relate to differences in preimplant PA pressures and differential SVR reduction in these CS phenotypes. These findings highlight the fact that although CS may be present in each, there are significant differences in treatment response in different CS etiologies. Furthermore, they shed light on the lack of benefit with IABP for AMI and the mounting evidence that IABP may stabilize many ADHF patients who deteriorate into CS.

Disclosures

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