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Coronary perfusion pressure and left ventricular hemodynamics as predictors of cardiovascular collapse following percutaneous coronary intervention☆

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ABSTRACT

Background/Purpose: Appropriate patient selection for mechanical circulatory support following percutaneous coronary intervention (PCI) remains a challenge. This study aims to evaluate the role of coronary perfusion pressure and other left ventricular hemodynamics to predict cardiovascular collapse following PCI.
Methods/Materials: We retrospectively analyzed all patients who underwent PCI for acute coronary syndrome (ACS) from 2003 to 2016. Coronary perfusion pressure was calculated for each patient and defined as the difference in mean arterial pressure and left ventricular end diastolic pressure (LVEDP). Logistic regression analysis was performed to determine predictor of composite outcome of in-hospital mortality, myocardial infarction (MI), congestive heart failure (CHF), and cardiogenic shock.
Results: Nine hundred twenty-two patients were analyzed. Two-hundred twenty-eight (25%) presented with ST-elevation MI (STEMI) while 694 (75%) underwent PCI for unstable angina or non-Q-wave MI. The mean LVEDP was significantly higher in the STEMI patients (24 ± 9 vs. 19 ± 8 mm Hg, $p < 0.05$) and perfusion pressure significantly lower (68 ± 24 vs. 74 ± 18 mm Hg, $p < 0.05$). Eighty-seven (9.4%) reached the composite endpoint, and there was no difference between the STEMI and Not-STEMI groups. Neither LVEDP nor coronary perfusion pressure was a predictor of the composite outcome following multivariable logistic regression analysis for either STEMI or Not-STEMI patients. Increasing age, chronic renal insufficiency (CRI), CHF, and low left ventricular ejection fraction were predictors of the composite outcome for Not-STEMI patients, whereas only history of cerebrovascular accident and CRI were predictors for STEMI patients.
Conclusions: In hemodynamically stable patients presenting with ACS, LVEDP and coronary perfusion pressure are not predictive of in-hospital cardiovascular collapse.
Summary: The authors retrospectively analyzed 922 patients from a single center who underwent percutaneous coronary intervention (PCI) for acute coronary syndromes to evaluate the role of coronary perfusion pressure and other left ventricular hemodynamics to predict cardiovascular collapse following PCI. They found that neither coronary perfusion pressure nor left ventricular end diastolic pressure was predictive of in-hospital cardiovascular collapse.

Abbreviations: ACS, acute coronary syndrome; CHF, congestive heart failure; CPP, coronary perfusion pressure; CRF, chronic renal failure; CRI, chronic renal insufficiency; ECMO, extracorporeal membrane oxygenation; IABP, intra-aortic balloon pump; LVEDP, left ventricular end diastolic pressure; MAP, mean arterial pressure; MCS, mechanical circulatory support; MI, myocardial infarction; NSTEMI, non-ST-elevation myocardial infarction; PCI, percutaneous coronary intervention; QWMI, Q wave myocardial infarction; STEMI, ST-elevation myocardial infarction.

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1. Introduction

Percutaneous mechanical circulatory support (MCS) has evolved dramatically since the intra-aortic balloon pump (IABP). There now exist a number of devices that may be employed directly in the catheterization laboratory for instances of cardiogenic shock and high-risk percutaneous coronary intervention (PCI). The list of devices includes the Impella, TandemHeart, and extracorporeal membrane oxygenation (ECMO) — all of which provide greater hemodynamic support compared to IABP. Despite studies demonstrating a lack of efficacy with the institution of IABP, the 2015 Expert Consensus of multiple societies advocates for the use of percutaneous MCS in cardiovascular care in order to reduce left ventricular stroke work and myocardial oxygen demand while maintaining systemic and coronary perfusion in the setting of cardiogenic shock or to provide hemodynamic support during complex cardiac procedures [1,2].

Cardiogenic shock is best defined as a spectrum of disease and can be classified as pre- or early shock, shock, and severe shock. Even still, once shock has developed, there are significant differences in patients with hemodynamic disturbances and hemometabolic disturbances. The hemodynamic, clinical, and vasoactive treatment parameters for each group have been described previously [3]. In the setting of acute coronary syndrome (ACS) and PCI, the rapid triage and early initiation of mechanical support is paramount to avoid "crashing onto support." However, patients presenting in pre- or early shock can often be difficult to diagnose and their clinical course unpredictable. It may be that identifying these patients early and treating with MCS results in the most benefit, but there is currently no reliable marker to predict the clinical course of a pre-shock patient, thus making patient selection very complicated.

Animal studies have demonstrated that a 40-mm Hg pressure gradient exists between coronary arterioles and venules, which is vital for adequate coronary perfusion [4]. In addition, hemodynamic literature also describes effective coronary perfusion pressure (CPP) as the difference in mean arterial pressure (MAP) and left ventricular end diastolic pressure (LVEDP), and it has been demonstrated as a predictor of mortality in patients presenting with cardiogenic shock [5,6]. Additionally, Alqarqaz et al. recently demonstrated a significant improvement in effective CPP in patients undergoing PCI with Impella MCS [7]. Assessing CPP in the setting of ACS at the time of PCI may help rapidly risk-stratify patients in the pre- or early shock scenario and aid in the decision to proceed with mechanical support. The objective of this study was to determine whether CPP impacted the development of the composite endpoint of cardiovascular collapse, in-hospital mortality, in-hospital myocardial infarction (MI), or need for repeat revascularization in stable patients presenting with ACS and undergoing PCI.

2. Materials and methods

This was a retrospective analysis of patients undergoing PCI at a single center in Washington, DC, from April 2003 to 2016 for a diagnosis of ACS, which included unstable angina, non-ST-elevation myocardial infarction (NSTEMI), and ST-elevation myocardial infarction (STEMI). Unstable angina was defined as typical chest pain of increasing frequency or intensity 2 weeks before index hospitalization that was refractory to medications and associated with dynamic ST-segment electrocardiographic abnormalities. NSTEMI was defined by the presence of typical chest pain or angina-equivalent symptoms in association with elevated troponin cardiac marker. STEMI was defined by the presence of typical chest pain or angina-equivalent symptoms in association with ST-segment elevation on presenting electrocardiogram or new left bundle-branch block.

Patients were only included in the analysis if there was recorded and documented measurement of LVEDP at any point during the index procedure. Finally, patients were excluded if there was a diagnosis of shock upon admission, the patient arrived to the catheterization laboratory with MCS such as IABP or vasoactive agents to maintain systolic pressure, or there was a need for emergent MCS prior to PCI.

LVEDP was obtained prior to or at the completion of index PCI. The end diastolic period was determined by corresponding electrocardiac monitoring and Q-wave timing. The LVEDP was recorded at end exhalation. CPP was then calculated by subtracting the LVEDP measurement from the patient's mean arterial systemic blood pressure measurement or MAP. MAP was determined by intra-aortic pressure monitoring and the value taken from left ventricular pullback. If the above time points were not available, intra-aortic MAP or non-invasive brachial cuff MAP were recorded and taken at the closest time point with respect to LVEDP measurement. The LVEDP and MAP were adjudicated by a single interventional cardiologist.

The primary outcome was a composite of in-hospital mortality, in-hospital MI or need for repeat revascularization, or the development of in-hospital cardiovascular collapse. Cardiovascular collapse was defined as the development of cardiogenic shock that was determined by ICD-9 and ICD-10 coding or the need for insertion of IABP or vasoactive agents, recurrent ischemia, and the development of congestive heart failure as determined by ICD-9 and ICD-10 coding using terms such as heart failure and pulmonary edema. MI was further characterized by Q wave MI (QWMI) if new Q waves deeper than 1 mm occurred in the 2 contiguous leads; otherwise non-QWMI was diagnosed. Chronic renal failure (CRF) was defined as serum creatinine >2.0 mg/dl. Stent thrombosis was defined in accordance with the Academic Research Consortium as definite or probable. The study was approved by the local institutional review board.

Table 1
Baseline demographics and characteristics of total cohort.

Variable	Not-STEMI ($n = 694$)	STEMI ($n = 227$)	Overall ($n = 921$)	Standardized difference	P value*
Male, *n* (%)	436 (63)	154 (68)	590 (64)	−0.106	0.999
Age, y ± SD	64 ± 12	59 ± 13	63 ± 12	0.383	<0.001
White, *n* (%)	340 (49)	91 (40)	431 (47)	0.173	0.999
Black, *n* (%)	294 (42)	101 (44)	395 (43)	−0.053	0.999
BMI ± SD	30 ± 6.8	30 ± 6.3	30 ± 6.6	−0.021	0.999
Hypertension, *n* (%)	567 (82)	168 (74)	735 (80)	0.189	0.165
Diabetes mellitus, *n* (%)	258 (37)	75 (33)	333 (36)	0.090	0.999
Hyperlipidemia, *n* (%)	513 (74)	118 (52)	631 (69)	0.471	<0.001
History of MI, *n* (%)	134 (20)	27 (12)	161 (18)	0.216	0.120
History of CAD, *n* (%)	248 (36)	41 (18)	289 (31)	0.409	0.003
History of CHF, *n* (%)	191 (28)	48 (22)	239 (26)	0.142	0.999
Renal insufficiency, *n* (%)	73 (10)	13 (5.7)	86 (9.4)	0.177	0.450
PVD, *n* (%)	64 (9.2)	9 (4.0)	73 (8.0)	0.212	0.180
Tobacco use, *n* (%)	346 (50)	137 (60)	483 (52)	−0.212	0.105
LVEF, % ± SD	48 ± 13	41 ± 12	46 ± 13	0.519	<0.001

* Bonferroni corrected p values ($0.05 * 15$).

Table 2
Baseline procedural characteristics and treatment of the total cohort.

Variable	Not-STEMI ($n = 898$)	STEMI ($n = 280$)	Overall ($n = 1178$)	Standardized difference	*P* value*
RCA, *n* (%)	309 (34)	105 (38)	414 (35)	−0.064	0.999
LAD, *n* (%)	307 (34)	122 (44)	429 (36)	−0.193	0.130
LCx, *n* (%)	252 (28)	48 (17)	300 (26)	0.263	0.009
SVG, *n* (%)	27 (3.0)	5 (1.8)	32 (2.7)	0.080	0.999
Proximal segment, *n* (%)	315 (36)	94 (34)	409 (35)	0.037	0.999
Bare metal stent, *n* (%)	71 (8.0)	31 (11)	102 (8.7)	−0.108	0.999
Drug-eluting stent, *n* (%)	720 (81)	212 (76)	932 (80)	0.118	0.999
Type C lesion, *n* (%)	269 (30)	101 (37)	370 (32)	−0.136	0.447
Angiographic success, *n* (%)	880 (98)	278 (99)	1158 (98)	−0.097	0.999

* Bonferroni corrected *p* values ($0.05 * 9$).

Table 3
Hemodynamic parameters at time of index procedure and PCI.

Variable	Not-STEMI ($n = 694$)	STEMI ($n = 227$)	Standardized difference	*P* value*
Heart rate, BPM ± SD	71 ± 14	72 ± 17	−0.473	<0.001
Systolic blood pressure, mm Hg ± SD	120 ± 39	108 ± 44	0.276	0.004
Diastolic blood pressure, mm Hg ± SD	68 ± 14	72 ± 17	−0.277	0.001
LVEDP, mm Hg ± SD	19 ± 8	24 ± 9	−0.595	<0.001
Mean arterial blood pressure, mm Hg ± SD	92 ± 16	93 ± 19	−0.037	0.999
Coronary perfusion pressure, mm Hg ± SD	74 ± 18	68 ± 24	0.246	0.005

* Bonferroni corrected *p* values ($0.05 * 6$).

Binary variables are reported by counts and percentages, while continuous variables are presented as mean ± SD. Binary data were compared by contingency table analysis (chi-square), and continuous data were compared by unpaired *t*-tests. Standardized differences were also calculated to indicate the degree of overlap of the distributions of the two groups. A standardized difference of 0.2 indicates about 15% non-overlap of the two distributions, 0.3 indicates about 23% non-overlap, and 0.5 indicates about 33% non-overlap. Hierarchical logistic regression (multiple vessels treated per patient) was used to compare procedural and treatment characteristics of the two groups. Univariate logistic regression analyses were performed to assess the impact of CPP and LVEDP on the composite outcome. Analyses were performed for Not-STEMI and STEMI groups separately. If a regression analysis was statistically significant, hemodynamic variables and other variables that potentially predict poor outcomes following ACS were added to assess improvement in model performance using likelihood ratio tests. A *p*-value of <0.05 was considered statistically significant. For comparisons of Not-STEMI and STEMI groups, a Bonferroni correction was made to control inflation of the Type I error rate due to multiplicity (0.05/number of tests). Statistical analyses were performed using SAS 9.4 (SAS Institute, Cary, NC) and Stata v15 (Stata Corp., College Station, TX).

3. Results

A total of 922 patients were analyzed. Two hundred twenty-eight (25%) presented with STEMI; 694 (75%) underwent PCI for unstable angina or non-QWMI (Not-STEMI). The average age was 63 ± 12 years. Sixty-four percent were male, 47% were Caucasian, and 43% were African-American. Eighty percent carried a diagnosis of hypertension, 36% had diabetes mellitus, 69% had hypercholesterolemia, 31% had a prior history of coronary artery disease, 9% a diagnosis of CRF, and 52% had a prior history of any tobacco use. The baseline characteristics and procedural characteristics for the two groups are listed in Tables 1 and 2, respectively.

The overall mean aortic systolic pressure was 125 ± 29 mm Hg with mean heart rate of 74 beats per minute. The mean LVEDP was significantly higher in the STEMI patients (24 ± 9 vs. 19 ± 8 mm Hg, $p < 0.05$) and perfusion pressure significantly lower (68 ± 24 vs. 74 ± 18 mm Hg, $p < 0.05$) compared to patients presenting with unstable angina or NSTEMI (Table 3).

Eighty-seven (9.4%) reached the composite endpoint of in-hospital mortality, MI, congestive heart failure, and cardiogenic shock, and there was no difference between the STEMI (10.1%) and Not-STEMI (9.2%) groups. Univariate logistic regression analyses are presented in Table 4. Lower CPP was associated with the composite endpoint for Not-STEMI patients (OR = 0.98, 95% CI = 0.96–0.99, $p = 0.011$). However, when adjusted for left ventricular ejection fraction, perfusion pressure was no longer statistically significant (OR = 0.99, 95% CI = 0.98–1.01, $p = 0.447$). No association of CPP and the composite endpoint was indicated for STEMI patients. LVEDP was not associated with the endpoint for either group. Increasing age, chronic renal insufficiency, congestive heart failure (CHF), heart rate, and low left ventricular ejection fraction were associated with the composite endpoint for Not-STEMI patients, whereas only history of cerebrovascular accident and chronic renal insufficiency (CRI) were predictive for STEMI patients (Fig. 1).

Table 4
Univariate logistic regressions of composite outcome as a function of CPP and LVEDP for not-STEMI and STEMI patients.

Variable	MI group	Odds ratio (95% CI)	*P* value
Coronary perfusion pressure	Not-STEMI	0.978 (0.962–0.995)	0.011
	STEMI	1.01 (0.987–1.034)	0.378
LVEDP	Not-STEMI	1.023 (0.99–1.057)	0.169
	STEMI	0.985 (0.937–1.037)	0.571

4. Discussion

Rapid identification of patients who may develop cardiogenic shock or cardiovascular collapse is paramount during MI. Various hemodynamic evaluations at the time of cardiac catheterization and PCI are often used to help make treatment decisions on mechanical support. There is much data on patients presenting with acute MI and cardiogenic shock, but the benefits of MCS are harder to prove and often futile in patients who are too sick. This study attempted to identify hemodynamic parameters that are helpful in diagnosing "early" or "pre-shock" conditions. The retrospective analysis of patients presenting with ACS revealed that those presenting with STEMI have higher LVEDP and lower CPP measurements. In a univariate model, low effective CPP was associated with worse in-hospital outcomes and increased

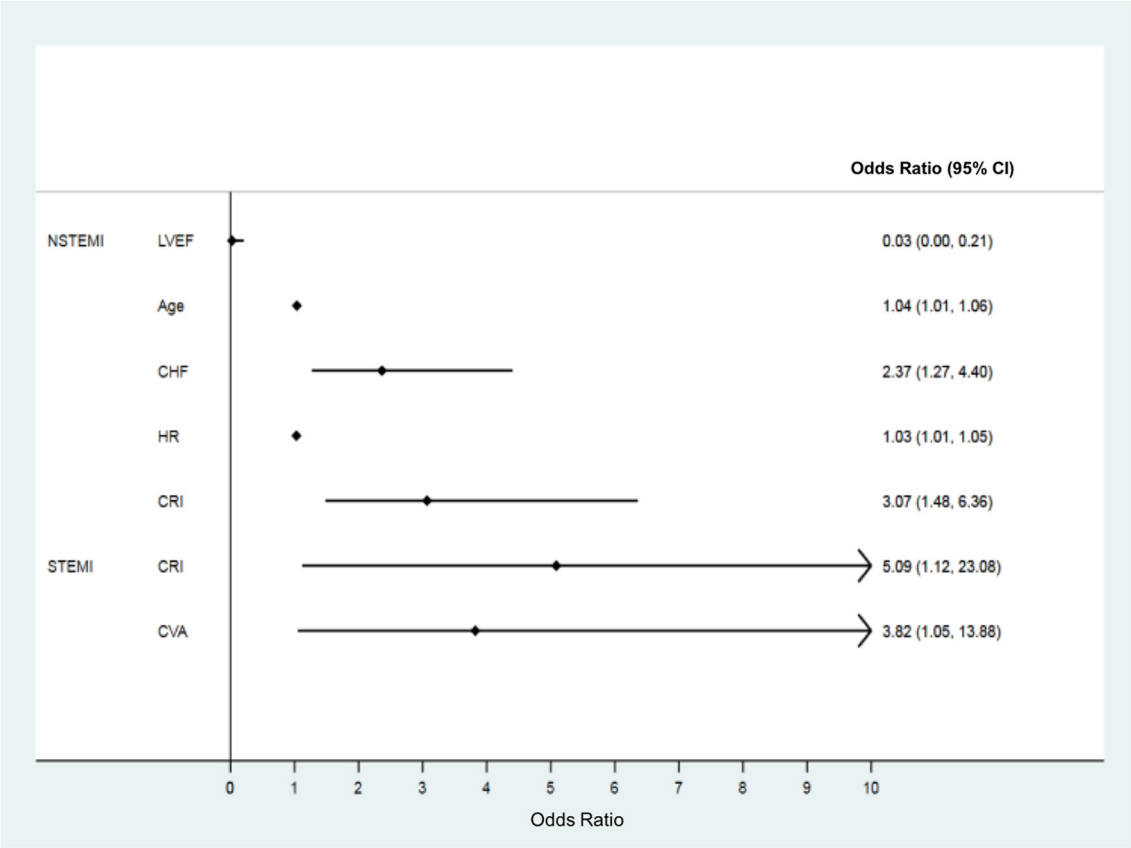

Fig. 1. Forest plot of significant predictors of outcome (in-hospital mortality, in-hospital MI, repeat revascularization or in-hospital cardiovascular collapse) for NSTEMI and STEMI patients.

likelihood of cardiovascular collapse in patients presenting with NSTEMI or unstable angina. LVEDP was not associated with the composite endpoint. In multivariate regression analysis, however, age, LVEF, CRI, prior cerebrovascular accident, and CHF were associated with the composite endpoint while CPP became insignificant.

The deterioration from pre-shock conditions to cardiogenic shock in the setting of acute MI has significant implications for morbidity and mortality. Despite early revascularization, it is estimated that 33% of patients with acute MI and cardiogenic shock will die during their hospitalization, and 30% of the survivors develop recurrent heart failure within the first year after discharge [8–10]. However, prior randomized controlled trials failed to demonstrate a clear benefit to the use of counter-pulsation devices during acute MI and cardiogenic shock [1,11]. Similarly, newer axial-flow devices, such as the Impella 2.5, failed to outperform the counter-pulsation balloon pump during PCI in high-risk individuals [12].

Despite a lack of data, the theoretical benefits of axial-flow devices in the setting of cardiogenic shock or high-risk PCI rely on improving 3 of 4 main hemodynamic perturbations during shock — namely, increased systemic perfusion, left ventricular unloading, and improved coronary perfusion [6]. There are currently ongoing trials to determine whether there is benefit to rapidly "unloading" the left ventricle in patients with acute MI and cardiogenic shock prior to revascularization with insertion of MCS devices. Another paradigm shift may include inserting such devices early on in the pre-shock state when there are hemodynamic changes only, before hemometabolic problems develop. The question then becomes how to rapidly and reliably identify those patients with high likelihood of developing shock and thus most likely to benefit from acute MCS.

Cardiac power output, a less commonly used indicator of myocardial contractility, which is calculated as (MAP × cardiac output/451) has been shown to be a strong marker of worsening clinical hemodynamics and a predictor of mortality in patients with advanced heart failure or cardiogenic shock [5,13,14]. Cardiac power output, however, requires a venous puncture, insertion of Swan-Ganz catheter, and measurement of cardiac output. A rapid test at the time of coronary angiography and an algorithm are needed to allow timely selection of patients at risk for hemodynamic deterioration. Prior studies of patients in cardiogenic shock have demonstrated that CPP is a predictor of in-hospital mortality.

In this particular study of patients with acute MI, CPP was a predictor of the predefined endpoint in univariate analysis but did not hold up in multivariate regression analysis. The lack of association may be due to the retrospective nature of the study and various time points at which hemodynamics measurements were obtained. In addition, the complexity of cardiogenic shock and multitude of variables that lead from hemodynamic changes to hemometabolic problems and eventual cardiovascular collapse suggests that a more complex prognostic model is necessary. Based on the results presented, increasing age, decreasing LVEF, and a history of CHF, CRI, or cerebrovascular accident may be variables to consider when deciding on acute mechanical circulatory support. In addition, neither LVEDP nor effective CPP is an independent predictor of the composite endpoint of mortality, ischemic events, and cardiovascular collapse in patients presenting with stable acute MI. Ultimately, further prospective studies are needed to determine key elements in predicting the development of cardiogenic shock.

4.1. Limitations

There are limitations to this study. First, the retrospective nature subjects it to the selection or information bias inherent to such a study design. For instance, missing data or variations in LVEDP measurement cannot be accounted for when collected retrospectively. Specifically, operators choose to measure LVEDP at different times throughout the

catheterization procedure depending on the clinical scenario. Similarly, the measurement of LVEDP and CPP is a single time point that varies throughout the cardiac procedure. It is likely more accurate to measure these parameters at several different times, as they are dynamic and subject to change based on medication administration, fluid administration, or PCI. The incidence of the outcome measured in this hemodynamically stable population was low and, therefore, the study is potentially underpowered for the hypothesis. Finally, cardiogenic shock or CHF as a clinical endpoint is difficult to catch in a retrospective manner and rely on accurate ICD diagnostic coding in this particular study.

5. Conclusions

In hemodynamically stable patients presenting with ACS, LVEDP and effective CPP are not predictive of in-hospital cardiovascular collapse. Routine measurement of LVEDP following PCI in stable patients may not be helpful to determine who will benefit from MCS. The quest for correlates to determine which patients in pre-shock conditions may develop cardiovascular collapse should be a subject for further prospective investigation.

Disclosures

Toby Rogers – Consultant: Medtronic.

Ron Waksman – Advisory Board: Abbott Vascular, Amgen, Boston Scientific, Medtronic, Philips Volcano, Pi-Cardia Ltd., Cardioset; Consultant: Abbott Vascular, Amgen, Biosensors, Biotronik, Boston Scientific, Medtronic, Philips Volcano, Pi-Cardia Ltd., Cardioset; Grant Support: Abbott Vascular, AstraZeneca, Biosensors, Biotronik, Boston Scientific, Chiesi; Speakers Bureau: AstraZeneca, Chiesi; Investor: MedAlliance.

All other authors – None.

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