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Left ventricular end-diastolic pressure affects measurement of fractional flow reserve

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Abstract

Background—Fractional flow reserve (FFR), the hyperemic ratio of distal (P_d) to proximal (P_a) coronary pressure, is used to identify the need for coronary revascularization. Changes in left ventricular end-diastolic pressure (LVEDP) might affect measurements of FFR.

Methods and Materials—LVEDP was recorded simultaneously with P_d and P_a during conventional FFR measurement as well as during additional infusion of nitroprusside. The relationship between LVEDP, P_a , and FFR was assessed using linear mixed models.

Results—Prospectively collected data for 528 cardiac cycles from 20 coronary arteries in 17 patients were analyzed. Baseline median P_a , P_d , FFR, and LVEDP were 73 mmHg, 49 mmHg, 0.69, and 18 mmHg, respectively. FFR < 0.80 was present in 14 arteries (70%). With nitroprusside median P_a , P_d , FFR, and LVEDP were 61 mmHg, 42 mmHg, 0.68, and 12 mmHg, respectively. In a multivariable model for the entire population LVEDP was positively associated with FFR such that FFR increased by 0.008 for every 1-mmHg increase in LVEDP ($\beta = 0.008$; $P < 0.001$), an association that was greater in obstructed arteries with FFR < 0.80 ($\beta = 0.01$; $P < 0.001$). P_a did not directly affect FFR in the multivariable model, but an interaction between LVEDP and P_a determined that LVEDP's effect on FFR is greater at lower P_a .

Conclusions—LVEDP was positively associated with FFR. The association was greater in obstructive disease (FFR < 0.80) and at lower P_a . These findings have implications for the use of FFR to guide revascularization in patients with heart failure.

Summary for Annotated Table of Contents—The impact of left ventricular diastolic pressure on measurement of fractional flow reserve (FFR) is not well described. We present a hemodynamic study of the issue, concluding that increasing left ventricular diastolic pressure can increase measurements of FFR, particularly in patients with FFR < 0.80 and lower blood pressure.

Keywords

fractional flow reserve; coronary artery disease; revascularization; heart failure

1. Introduction

More than 16.3 million Americans have coronary artery disease (CAD), and more than 6.6 million have heart failure [1]. CAD and heart failure may co-exist in patients who present with chest discomfort. Coronary angiography may be helpful in such patients, but the functional significance of angiographically identified coronary stenoses is sometimes

uncertain. Invasively measured fractional flow reserve (FFR) is currently the gold standard for assessment of the functional significance of CAD and is used to identify the need for coronary revascularization [2–6]. Calculation of myocardial FFR by the originally derived equation requires the subtraction of post-arteriolar coronary pressure (P_v) [3,7]. However, the inclusion of P_v may be unnecessary [8–11], and recent, landmark clinical trials of the clinical value of FFR have ignored P_v [12,13].

High left ventricular end-diastolic pressure (LVEDP) may impair myocardial blood flow by increasing coronary zero flow pressure [14], affecting measurement of FFR. Previous studies have not included substantial numbers of patients with significant left ventricular (LV) dysfunction [4,8–13]. Thus, the impact of changes in LVEDP on FFR measurement is largely unknown. We examined the relationship between changes in LVEDP and FFR. We hypothesized that LVEDP would be positively associated with FFR, and that the association would be greater in obstructive disease ($FFR < 0.80$).

2. Methods

The study protocol was approved by the local Institutional Review Board, and all patients provided informed consent. Procedures were performed in the cardiac catheterization laboratories at the Medical University of South Carolina. The study population consisted of 17 of 37 consenting patients who had at least one vessel with at least intermediate (40%) disease in whom adequate FFR tracings could be obtained both before and during nitroprusside infusion. FFR measurement was performed according to recommended standards [15,16] using pressure wires (RadiMedical Systems, Inc., Uppsala, Sweden), which were externally calibrated. Guiding catheters were purged of contrast material and filled with saline, and pressure tracings from the guiding catheter and pressure wire were equalized. Maximal hyperemia was induced by intravenous infusion of adenosine at 140 $\mu\text{g}/\text{kg}/\text{min}$. Using a pigtail catheter placed from a second arterial access site into the left ventricle, LVEDP was recorded simultaneously with P_d and P_a . To reduce LVEDP sodium nitroprusside was then infused intravenously at 1 $\mu\text{g}/\text{kg}/\text{min}$ during continued adenosine infusion. The nitroprusside infusion was rapidly titrated in increments of 0.5 to 1 $\mu\text{g}/\text{kg}/\text{min}$ until the LVEDP was reduced to <10 mmHg or the systolic systemic arterial pressure was reduced to <80 mmHg. FFR was measured as the ratio of mean P_d to mean P_a during infusion of adenosine at baseline as well as during infusion of nitroprusside. Attention was paid to avoid pressure damping related to deep engagement of the guiding catheter. Wire pullback was used to assess for signal drift, and repeated measurement was completed as needed.

Simultaneously recorded pressures were reviewed offline by two investigators at scales of 200 mmHg, 100 mmHg, and 40 mmHg to facilitate accurate assessment of both systolic and diastolic pressures, which were assessed on a beat-to-beat basis starting from the R wave on the co-registered electrocardiographic tracing. Premature ventricular beats and post-premature beats were excluded from analysis. Mean P_d and P_a were calculated for every cardiac cycle by doubling the minimal diastolic pressure, adding the systolic pressure, and dividing by 3. FFR was calculated as P_d/P_a . $FFR < 0.80$ was considered to be physiologically obstructive. LV diastolic pressures were measured at end-diastole, which was defined electrocardiographically by the onset of the next cycle's R wave.

The relationships between LVEDP, P_a , and FFR were examined using linear mixed models including a random subject effect to account for correlation among measurements on the same subject. Compound symmetry and variance component covariance structures were considered, and the latter was selected due to better fit. Univariate models as well as a multivariable model including LVEDP, P_a , and the interaction between LVEDP and P_a as

covariates were examined. Sub-group analysis was completed for patients with FFR < 0.80. All analyses were done in SAS version 9.2 (SAS Institute, Cary, NC).

3. Results

Patient characteristics are summarized in Table 1. A total of 20 coronary arteries were interrogated, and 528 cardiac cycles were analyzed. In 10 of these vessels, the infusion was stopped as LVEDP decreased to 10 mm Hg while in the remaining 10 vessels, the infusion was stopped for SBP 80 mmHg. In two patients the infusion was stopped due to hypotension, and these patients were not included in the analysis. During baseline FFR measurement the median (interquartile range) P_a , P_d , FFR, and LVEDP for all cardiac cycles in all arteries were 73 (63–85) mmHg, 49 (45–57) mmHg, 0.69 (0.60–0.84), and 18 (13–24) mmHg, respectively. FFR averaged for all beats in each artery was < 0.80 in 14 of 20 arteries (70%). During nitroprusside infusion the median P_a , P_d , FFR, and LVEDP were 61 (58–65) mmHg, 42 (38–49) mmHg, 0.68 (0.64–0.83), and 12 (8–18) mmHg, respectively. Aggregate arterial characteristics at angiography are summarized in Table 2, while individual arterial hemodynamic characteristics before and during nitroprusside infusion are summarized in Table 3. Beat-to-beat associations of LVEDP with FFR in representative arteries are shown in Fig. 1.

Results of the regression models are summarized in Table 4 and Fig. 2. For the entire population both LVEDP and P_a were significantly positively associated with FFR in the univariate model. The univariate association between LVEDP and FFR was such that FFR increased by 0.003 for every 1-mmHg increase in LVEDP. The univariate association between P_a and FFR was such that FFR increased by 0.001 for every 1-mmHg increase in P_a . In the multivariable model the association between LVEDP and FFR was such that FFR increased by 0.008 for every 1-mmHg increase in LVEDP at the mean P_a for the entire population. After controlling for LVEDP, P_a did not directly affect FFR in the multivariable model. The multivariable model included a significant ($P = 0.011$) interaction between LVEDP and P_a such that the magnitude of the association of LVEDP with FFR was dependent on P_a . The effect of LVEDP on FFR was greater when P_a was lower (Fig. 2B).

For the sub-group with FFR < 0.80 both LVEDP and P_a were significantly positively associated with FFR in the univariate model. The univariate association between LVEDP and FFR was such that FFR increased by 0.004 for every 1-mmHg increase in LVEDP. The univariate association between P_a and FFR was such that FFR increased by 0.001 for every 1-mmHg increase in P_a . In the multivariable model the association between LVEDP and FFR was such that FFR increased by 0.012 for every 1-mmHg increase in LVEDP at the mean P_a for the entire population. Again, after controlling for LVEDP, P_a did not directly affect FFR in the multivariable model. Again, the multivariable model included a significant ($P = 0.002$) interaction between LVEDP and P_a such that the magnitude of the association of LVEDP with FFR was dependent on P_a . The affect of LVEDP on FFR was greater when P_a was lower (Fig. 2 C).

4. Discussion

We found in this prospective study of 528 cardiac cycles and 20 coronary arteries that there was an association between LVEDP and FFR measurements, an association which was greatest in patients with obstructive CAD (FFR < 0.80) and lower blood pressure. These data demonstrate for the first time to our knowledge that LVEDP is a determinant of conventionally measured FFR.

Landmark trials [4,12,13] of FFR were designed to enroll patients with normal LV systolic function and have not included assessments of left ventricular diastolic pressure in their methods. To our knowledge, no prior studies assessing the clinical utility of FFR have reported measurements of LVEDP. Our data suggest that, compared to FFR at normal LVEDP, FFR at higher LVEDP is increased. Thus, the sensitivity of $\text{FFR} < 0.80$ for the identification of obstructive CAD may be reduced when LVEDP is high. These findings may have implications for the use of FFR to guide revascularization in patients with heart failure and CAD.

Our results add to previously published data. A study of myocardial blood flow and LVEDP reported that high LVEDP directly impedes myocardial perfusion [14]. By this mechanism it is possible that high LVEDP functionally creates microvascular disease, which is known to cause inflation of measured FFR [17]. An abstract [18] presented at the American Heart Association Scientific Sessions in 2010 reported by multivariable analysis a strong trend for LVEDP change to predict FFR change [18]. The authors concluded that underestimation of the severity of CAD might be anticipated in cases of significantly increased LVEDP.

We also found that changes in P_a during nitroprusside infusion were not associated with changes in FFR after controlling for LVEDP, a finding that is consistent with previously reported data from a study of 10 coronary stenoses assessed using FFR measurement with intracoronary adenosine as well as additional, intravenous infusion of nitroprusside [19]. This study included patients with normal LV systolic function (and presumably normal LVEDP), and measured FFR did not change despite substantial reductions in systolic blood pressure with nitroprusside infusion.

Our final multivariable model included a significant interaction between LVEDP and P_a , suggesting that the magnitude of the association of LVEDP with FFR is dependent on P_a and that the effect of LVEDP on FFR is greater when P_a is lower. Conversely, higher P_a is expected to reduce the effect of LVEDP on FFR. Thus, while P_a did not affect FFR directly, P_a did affect FFR indirectly by way of its effect on the association of LVEDP with FFR.

Our study has several limitations. First, it is a small study of only 17 patients. Second, it is possible that observed decreases in FFR during infusion of nitroprusside were related in part to additional hyperemia (reduction in vascular resistance) as opposed to reduction in LVEDP alone; however, studies have shown that intravenous adenosine infusion at $140 \mu\text{g/kg/min}$ induces maximal hyperemia [20,21]. Furthermore, in the study referenced above [19], FFR was not changed by the infusion of nitroprusside. Third, we cannot exclude the possibility that the observed association between LVEDP and FFR might be a reflection of changes in coronary venous pressure (an unmeasured variable in our study).

Maximal hyperemia has been called the “last remaining barrier” to routine, clinical use of FFR in the cardiac catheterization laboratory [22]. The results of this study and previous studies [14,23,24] combine to suggest that elevated LVEDP may be another confounder of FFR measurement and that further study is needed before a measured FFR of slightly > 0.80 can be used to confidently exclude physiologically significant CAD in patients with substantially elevated LVEDP. Given the high and increasing prevalence of CAD with co-morbid heart failure such research is warranted.

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Fig. 1.

Beat-to-beat Association of Left Ventricular End-Diastolic Pressure with Fractional Flow Reserve in Representative Arteries with Obstructive vs. Non-Obstructive Disease. (A) Each point represents a single cardiac cycle from a coronary artery with obstructive disease (mean fractional flow reserve = 0.76 for all adenosine-only beats). (B) Each point represents a single cardiac cycle from a coronary artery with non-obstructive disease (mean fractional flow reserve = 0.94 for all adenosine-only beats).

Fig. 2.

Estimated Regression Lines. (A) Estimated regression lines for the univariate models of the association between LVEDP and FFR for all subjects vs. subjects with baseline mean FFR < 0.80 for adenosine-only beats ($P < 0.001$ for both). (B) Estimated regression lines for the multivariable model of the association between LVEDP and FFR when P_a is held constant at either 40 or 100 mmHg for all subjects ($P = 0.011$). (C) Estimated regression lines for the multivariable model of the association between LVEDP and FFR when P_a is held constant at either 40 or 100 mmHg for subjects with FFR < 0.80 ($P = 0.002$).

Table 1

Patient Characteristics.

Characteristic	
Age (years $\pm$ SD)	65.3 $\pm$ 11.5
Male (n (%))	14 (82.4%)
ST-elevation Myocardial Infarction (n (%))	0 (0%)
Non-ST-elevation Myocardial Infarction (n (%))	2 (11.7%)
Unstable Angina (n (%))	8 (47.1%)
Stable Angina (n (%))	7 (41.2%)
Left Ventricular Ejection Fraction (% $\pm$ SD)	57.5 $\pm$ 14.6
Hypertension (n (%))	16 (94.1%)
Diabetes (n (%))	6 (35.3%)
Hemoglobin (g/dL $\pm$ SD)	13.3 $\pm$ 1.9
Prior Q-wave Myocardial Infarction in Studied Territory (n (%))	1 (5.9%)

Diabetes was considered present in the setting of treatment for diabetes or hemoglobin A1c > 7%. Q-wave myocardial infarction was defined electrocardiographically by the presence of pathologic Q waves in 2 leads corresponding to the target artery's distribution.

Table 2

Aggregate Coronary Arterial Characteristics at Angiography.

Characteristic	
Left Main Coronary Artery (n (%))	1 (5%)
Left Anterior Descending Coronary Artery (n (%))	7 (35%)
Left Circumflex Coronary Artery (n (%))	8 (40%)
Ramus Intermedius Coronary Artery (n (%))	1 (5%)
Right Coronary Artery (n (%))	3 (15%)
Mean Proximal Reference Diameter (mm $\pm$ SD)	2.9 $\pm$ 0.5
Mean Distal Reference Diameter (mm $\pm$ SD)	2.6 $\pm$ 0.3
Mean % stenosis ((% $\pm$ SD), range)	71 $\pm$ 14, 50-95
Mean Lesion Length (mm $\pm$ SD)	20.0 $\pm$ 16.2
Lesion Type A (n (%))	3 (15%)
Lesion Type B (n (%))	9 (45%)
Lesion Type C (n (%))	8 (40%)
Mild Calcification (n (%))	6 (30%)
Moderate-severe Calcification (n (%))	3 (15%)
Tortuosity (n (%))	3 (15%)
Serial Lesions (n (%))	3 (15%)

Table 3
Individual Coronary Arterial Hemodynamic Characteristics Before and During Nitroprusside Infusion.

Artery	Before Nitroprusside					During Nitroprusside				
	Beats	P _a	P _d	FFR	LVEDP	Beats	P _a	P _d	FFR	LVEDP
LCx	14	86	41	0.48	21	14	66	26	0.39	8
LCx	12	58	46	0.79	20	14	48	34	0.71	15
LCx	12	76	44	0.58	11	13	61	42	0.68	10
LAD	12	70	49	0.70	9	14	71	54	0.76	12
LIMA-LAD	12	72	51	0.70	24	15	63	43	0.68	24
LCx	13	73	48	0.66	22	14	60	42	0.70	20
RI	12	94	94	1.00	21	11	99	92	0.93	22
LAD	14	59	34	0.58	17	14	58	38	0.66	22
LCx	14	64	56	0.87	8	13	57	48	0.83	6
RCA	18	94	58	0.62	20	19	75	48	0.64	13
LAD	15	71	45	0.64	14	19	61	39	0.64	10
LCx	13	60	50	0.83	16	12	62	54	0.87	9
LAD	11	73	56	0.76	25	16	60	41	0.67	11
LAD	14	77	51	0.66	7	16	65	43	0.67	6
LCx	14	88	19	0.22	24	13	64	13	0.21	8
LAD	15	62	59	0.96	14	14	43	39	0.92	4
LCx	8	66	46	0.70	18	7	58	41	0.70	16
LAD	11	83	78	0.94	22	8	52	49	0.95	9
LCx	12	86	45	0.52	28	11	60	29	0.48	10
LCx	12	60	59	0.97	28	13	54	52	0.98	26

Hemodynamic parameters are means for all cardiac cycles obtained in each condition. Pressures are reported in millimeters of mercury. P_a = proximal coronary artery pressure. P_d=distal coronary artery pressure. FFR=fractional flow reserve. LVEDP = left ventricular end-diastolic pressure. LCx = left circumflex coronary artery. LAD = left anterior descending coronary artery. LIMA-LAD = left internal mammary artery graft to the left anterior descending coronary artery. RI = ramus interemedius. RCA = right coronary artery.

Table 4

Results of Regression Models.

A. Results for the Entire Population				
Model	Parameter	Beta	SE	P value
Univariate for LVEDP	Intercept	0.6539	0.0425	<0.001
	LVEDP	0.00336	0.00025	<0.001
Univariate for P_a	Intercept	0.6303	0.0456	<0.001
	P_a	0.00113	0.00022	<0.001
Multivariable for LVEDP and P_a	Intercept	0.6638	0.0430	<0.001
	LVEDP	0.00769	0.00131	<0.001
	P_a	-0.00038	0.00037	0.315
	LVEDP* P_a	-0.00005	0.00002	0.011
B. Results for Patients with FFR < 0.80				
Model	Parameter	Beta	SE	P value
Univariate for LVEDP	Intercept	0.5496	0.0389	<0.001
	LVEDP	0.00403	0.00029	<0.001
Univariate for P_a	Intercept	0.5375	0.0438	<0.001
	P_a	0.00110	0.00028	<0.001
Multivariable for LVEDP and P_a	Intercept	0.5653	0.0430	<0.001
	LVEDP	0.0115	0.0019	<0.001
	P_a	-0.00038	0.00051	0.199
	LVEDP* P_a	-0.00008	0.00003	0.002

Abbreviations as in Table 3.