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Original Article

Combined atrioventricular longitudinal strain rate during isovolumic contraction predicts pulmonary capillary wedge pressure in patients with systolic dysfunction

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Abstract: Background: Reportedly, mitral annular velocities derived by tissue Doppler imaging (TDI)-during isovolumic contraction (IVV) can predict pulmonary capillary wedge pressure (PCWP) in heart failure patients with depressed ejection fraction (EF). We investigated the use of color TDI-derived left atrial (LA) and left ventricular (LV) longitudinal strain rate (SR) during isovolumic contraction (IC) to predict the invasively measured PCWP. Methods and results: Forty patients referred with symptoms of heart failure were prospectively studied [age: 56±8 years, 12 (30%) females, and mean LVEF: 51±14%]. PCWP was measured invasively immediately after echocardiography. Mitral annular IVV was measured for all patients and SR during the IC and ejection were measured for the LV (LVSR-IC, LVSR-Ej) as well as the LA (LASR-IC, and LASR-Ej). Atrioventricular SR during IC and Ej (AVSR-IC, AVSR-Ej) was calculated as the sum of the LV and LA values. Patients were classified and compared based on their EF into 19 (49%) with EF≥55%, and 21 (51%) with EF<55%. No significant differences were noted for age, sex, risk factors, and medications between both patients with EF≥55% and EF<55%. Compared to EF≥55%, patients with EF<55% had lower IVV (4.63±1.2 vs. 7.01±1.9 cm/s, P<0.001), LVSR-Ej (1±0.3 vs. 1.2±0.2, P=0.03), LASR-IC (1.3±0.6 vs. 1.9±1, P=0.03), LASR-Ej (1.5±0.5 vs. 2.6±1.3 s^{-1}, P=0.001), AVSR-IC (2±0.8 vs. 2.7±1.06 s^{-1}, P=0.023), and AVSR-Ej (2.5±0.6 vs. 3.9±1.1 s^{-1}, <0.001). LVSR-IC, LVSR-Ej, LASR-IC, AVSR-IC, and IVV correlated with PCWP in only in EF<55%, with the strongest correlation noted for AVSR-IC (r=-0.72, <0.001). Other correlates with PCWP in EF<55% were E/e' and left atrial volume (r=0.47, 0.7, P=0.04, 0.001; respectively). Multivariate regression revealed that in patients with EF<55% AVSR-IC was the only independent predictor of PCWP. Finally, IVV correlated with LVSR-IC and LASR-IC and this correlation became strongest with AVSR-IC (r=0.77, 0.001). Conclusion: The combined LV and LA longitudinal SR during IC as represented by AVSR-IC showed a strong correlation with PCWP in patients with depressed EF. The correlation between mitral annular IVV and PCWP in those patients can be a product of this combination and may a function of atrioventricular mechanical coupling.

Keywords: Isovolumic contraction, longitudinal strain rate, heart failure, pulmonary capillary wedge pressure

Introduction

Heart failure (HF) is a complex clinical condition that can result from abnormal systolic or diastolic functions. Regardless of the type of heart failure, it can be hemodynamically significant causing elevation of pulmonary capillary wedge pressures (PCWP) [1]. Invasive cardiac catheterization remains the reference method for cardiac pressure measurement including PCWP. In clinical practice, echocardiography is considered the standard non-invasive method for the assessment of left ventricular filling pressures and most of the echocardiographic variables used are derived from the diastolic phase as parameters derived from the systolic phase, such as the ejection fraction EF, are load-dependent and do not correlate with inva-

sive pressure measurements [2]. The isovolumic contraction (IC) phase, on the other hand, is relatively load-independent, and reportedly variables derived from the IC such as tissue Doppler (TDI)-derived mitral annular velocity (IVV) have been reportedly useful for the assessment of LV functions [3-6]. IC is a dynamic part of the cardiac cycle during which subendocardial fiber shortening is offset by subepicardial sheet extension and negative angle formation to keep the cavity within isovolumetric constraints [7]. IC seems to be linked to more efficient ejection and, deformations that occur during IC are thought to be stored in memory to be released during isovolumic relaxation participating in diastolic suction pressure formation [8]. Recently, tissue Doppler-derived velocity (IVV) and acceleration during IC are suggested as load-independent indicators of myocardial systolic function and are reportedly predictors of VO_2max in patients with systolic dysfunction [5]. Moreover, we recently reported that IVV can also predict PCWP in patients with systolic dysfunction and not in patients with normal systolic functions [4]. However, deformations during isovolumic contraction also occur in the atrium [9], and whether or not the mitral annular velocities and their association with cardiac function is the result of the combined forces of the left ventricle and the left atrium remain unknown. We hypothesized that the predictive ability of the longitudinal deformations during IC combined from both the LA and the LV will be a better predictor of PCWP than either alone.

Methods

In a prospective study protocol, forty consecutive patients referred to our echocardiography department to assess myocardial functions because of heart failure symptoms were studied. All patients had an invasive right heart catheterization and PCWP was invasively measured immediately after echocardiography. Patients were excluded if the age was less than 18 years, pregnant, had more moderate or severe mitral valve regurgitation or mitral valve stenosis (which render mitral annular measurements unreliable), if they had poor echocardiographic windows, or refused to participate in the study.

Patients were then classified and compared according to their systolic function, defined by EF, into patients with EF≥55%, and patients with EF<55%.

The study protocol was approved by the Ain Shams University institutional review board (IRB) with approval number FWA000017585. All patients provided informed consent for all procedures and the authors had full access to the data and take full responsibility for their integrity.

Echocardiographic examination

Echocardiography was performed using commercially available systems equipped with a 2.5 megahertz phased array transducer. Digital routine 2-D and color tissue Doppler cine loop images from three consecutive cardiac cycles as confirmed by electrocardiography were captured at end-expiratory apnea from the standard apical views at depths of 12-20 cm. The sector was optimized so that it allows for complete visualization of the myocardium with the maximum frame rate possible. Gain settings were optimized for appropriate endocardial border definition. The LV-EF was averaged from apical 2-chamber and 4-chamber images using the biplane Simpson's technique, while Left atrial volume (LAV) was calculated from 2-chamber and 4-chamber views using the biplane prolate ellipse method.

Pulsed-wave doppler examination

Trans-mitral flow velocities were obtained using pulsed wave Doppler imaging at the tip of the mitral leaflets, and color tissue Doppler imaging (TDI)-derived mitral annular velocities were obtained from the apical 4-chamber view. E-wave velocity was defined as the peak velocity of the early diastolic mitral flow wave and the atrial contraction A wave velocity was defined as the peak velocity of the late mitral flow wave. TDI-derived mitral annular early diastolic velocity (e'), as well as IC velocity (IVV), were obtained and averaged from the septal, lateral, anterior, and inferior mitral annular positions (**Figure 1**). All measurements made during IC were taken from the positive wave only. The LV filling pressures were assessed using the E/A and E/e' ratios.

Color tissue Doppler deformation studies

Color TDI was used to derive myocardial longitudinal deformations of the LV and the LA from

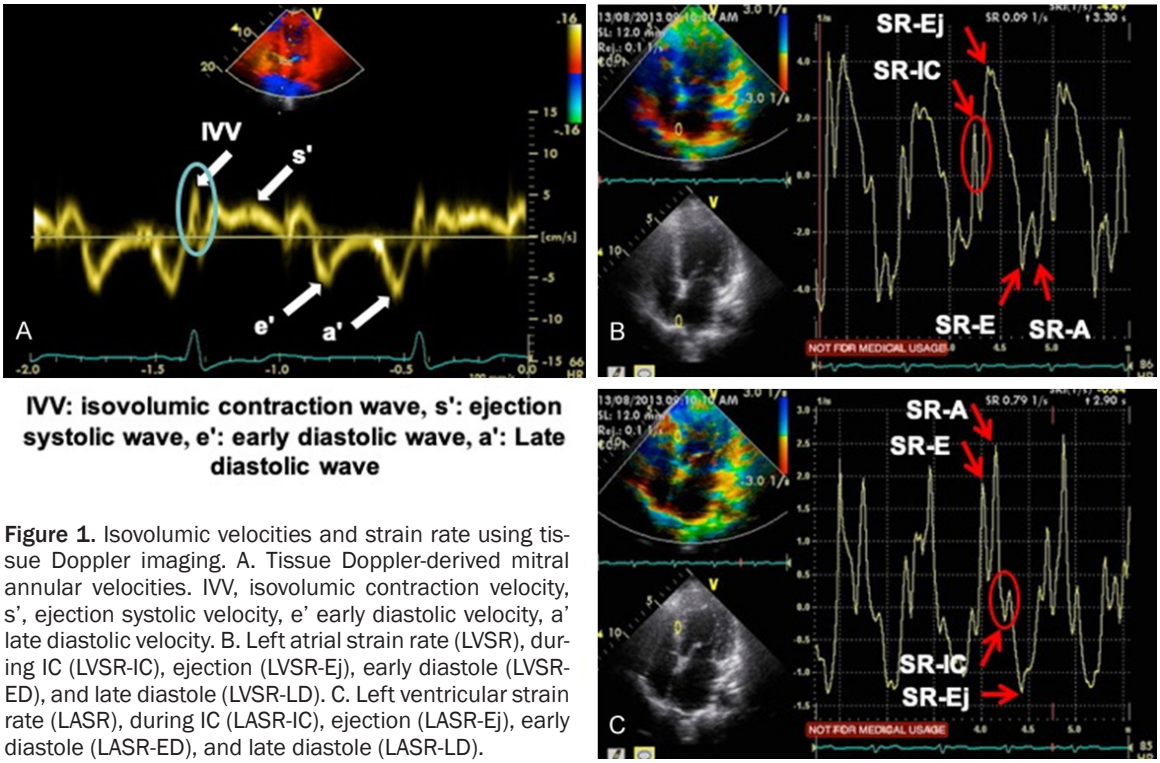

Figure 1. Isovolumic velocities and strain rate using tissue Doppler imaging. A. Tissue Doppler-derived mitral annular velocities. IVV, isovolumic contraction velocity, s', ejection systolic velocity, e' early diastolic velocity, a' late diastolic velocity. B. Left atrial strain rate (LVSR), during IC (LVSR-IC), ejection (LVSR-Ej), early diastole (LVSR-ED), and late diastole (LVSR-LD). C. Left ventricular strain rate (LASR), during IC (LASR-IC), ejection (LASR-Ej), early diastole (LASR-ED), and late diastole (LASR-LD).

the apical 4-chamber, 2-chamber, and 3 chamber views (**Figure 1B** and **1C**). Peak longitudinal systolic LVSR and LASR during IC and ejection (Ej) were measured. LVSR-IC and LVSR-Ej were measured and averaged from 12 basal and mid segments corresponding to 6 LV walls in apical views, while mean LASR-IC and LASR-Ej were measured and averaged from the lateral, septal, anterior and inferior LA walls, corresponding to the mitral annular positions from which IVV was measured as previously described. Atrioventricular SR during IC (AVSR-IC) and Ej (AVSR-Ej) was measured by summing the values of LV and LA.

Cardiac catheterization

All catheter measurements were done by an investigator blinded to the echocardiographic data. All patients underwent right heart catheterization for hemodynamic measurements using a fluid-directed balloon-tipped catheter. Fluoroscopically verified mean PCWP was obtained at end-expiration with the zero-level set at the midaxillary line and represent the average of 5 cardiac cycles.

Statistical analysis

Continuous data were expressed as mean ± SD and categorical data was expressed as number (%). The independent sample t-test was used to compare the mean values of different groups. Linear regression expressed as Pearson correlation coefficient was used for correlation analyses between the non-invasively measured echocardiographic variables and the invasively measured PCWP. Multivariate regression analysis based on stepwise selection was initiated for the assessment of the independent determinants of PCWP. $P<0.05$ was considered the limit for statistical significance for all tests. All statistical analyses were performed using commercially available software (SPSS version 21.0; SPSS, Inc, Chicago, IL, USA).

Results

Initially, 52 patients were screened during the study period, 5 patients were excluded due to moderate or severe mitral regurgitation (2 rheumatic and 3 due to tethered leaflets), 3 patients were excluded due to moderate or severe rheumatic mitral stenosis, and 4 patients were excluded due to poor echocardiographic windows. Accordingly, the final study cohort included 40 patients. The mean age was 56.8±25 years and 12 (30%) patients were females. Based on EF patients were classified into 19 patients (%49) with EF≥55%, and 21 patients (51%) with EF<55%. **Table 1** summarizes baseline clini-

Table 1. Demographics, clinical and echocardiographic parameters overall and in subgroups

	All (n=40)	EF≥55% (n=19)	EF<55% (n=21)	*P*-value
Age, year	56.8±25	57.6±8.9	54±7.7	0.224
Sex (female), n (%)	12 (30%)	7 (37%)	5 (24%)	0.728
New York Heart Association Functional class	2.1±0.58	1.94±0.56	2.3±0.57	0.088
Systolic blood pressure, mmHgg	144.4±36.6	147.5±34	141.3±40	0.637
Diastolic blood pressure, mmHg	86.22±22.865	87.4±13.1	85±30	0.768
Mean blood pressure, mmHg	111.16±27.9	112.2±23	110.1±33.2	0.837
Risk Factors				
Diabetes mellites, n (%)	10 (25%)	5 (26%)	5 (24%)	0.915
hypertension, n (%)	14 (35%)	8 (42%)	6 (29%)	0.897
hyperlipidemia, n (%)	2 (5%)	1 (5%)	1 (5%)	0.683
Smoker, n (%)	8 (20%)	5 (26%)	3 (14%)	0.907
Medications				
Aspirin, n (%)	13 (33%)	7 (37%)	6 (29%)	0.873
Clopedogrel, n (%)	4 (10%)	1 (5%)	3 (14%)	0.222
Renin-Angiotensin-Aldosterone Antagonists, n (%)	6 (15%)	2 (11%)	4 (19%)	0.432
B-Blockers, n (%)	11 (28%)	5 (26%)	6 (29%)	0.621
Spironolactone, n (%)	2 (5%)	0 (0%)	2 (10%)	0.247
Digoxin, n (%)	2 (5%)	1 (5%)	1 (5%)	0.683
Furosemide, n (%)	6 (15%)	2 (11%)	4 (19%)	0.432
Nitrates, n (%)	11 (28%)	5 (26%)	6 (29%)	0.621
Statins, n (%)	4 (10%)	2 (11%)	2 (10%)	0.997
Mitral flow E-wave velocity, cm/s	82.7±28.5	70.7±17.3	94.3±32	0.008
Mitral flow A-wave velocity, cm/s	78.1±26.7	81.3±19.1	74.6±33	0.462
E-wave Deceleration time, msec	218.2±104	234.6±62.4	209±130	0.452
Average mitral annular e' velocity, cm/s	7.4±2.2	7.7±2.2	7.1±2.2	0.369
Average mitral annular s' velocity, cm/s	7.1±1.9	8.2±1.5	6.1±1.7	<0.001
Tricuspid regurgitation pressure gradient, mmHg	25.1±13.04	22.5±9.8	27.6±15.6	0.346
Left atrial volume, ml	68.3±25.6	61.3±19.8	74.6±28.9	0.119
Left ventricular end diastolic volume, ml	131.7±53	100±18.8	158.6±58	<0.001
Left ventricular end systolic volume, ml	69.5±50.6	37±9.1	97.1±55.2	<0.001
Ejection fraction, %	51±14	62.8±4.4	41.2±9.3	<0.001
E/A ratio	1.18±0.67	0.96±0.48	1.46±0.77	0.033
E/e' ratio	12.46±6.59	3.7±0.9	7.5±1.8	0.016
Isovolumic contraction velocity (IVV), cm/s	6.01±2.4	7.01±1.9	4.63±1.2	<0.001
Pulmonary Capillary Wedge Pressure (PCWP), mmHg	20.35±7.85	16.2±4.8	23.9±8.5	0.002
Pulmonary artery systolic pressure, mmHg	41.38±11.87	37.9±1.7	44.4±12.2	0.06
Pulmonary artery diastolic pressure, mmHg	23.14±7.43	20.1±5.6	25.9±7.9	0.016
Pulmonary artery mean pressure, mmHg	28.69±8.774	25±5.5	31.8±9.9	0.02
LVSR-IC, s^{-1}	0.76±0.25	0.84±0.28	0.68±0.2	0.08
LASR-IC, s^{-1}	1.6±0.86	1.9±1	1.3±0.6	0.03
AVSR-IC, s^{-1}	2.3±0.98	2.7±1.06	2±0.8	0.023
LVSR-Ej, s^{-1}	1.11±0.28	1.2±0.2	1±0.3	0.03
LASR-Ej, s^{-1}	2.06±1.08	2.6±1.3	1.5±0.5	0.001
AVSR-Ej, s^{-1}	3.17±1.18	3.9±1.1	2.5±0.6	<0.001

Parameters expressed as mean ± SD or number (%). LVSR-IC, left ventricular longitudinal strain rate during isovolumic contraction, LASR-IC, left atrial longitudinal strain rate during isovolumic contraction, AVSR-IC, atrio-ventricular longitudinal strain rate during isovolumic contraction, LVSR-Ej, left ventricular longitudinal strain rate during ejection, LASR-Ej, left atrial longitudinal strain rate during ejection, AVSR-Ej, atrio-ventricular longitudinal strain rate during ejection.

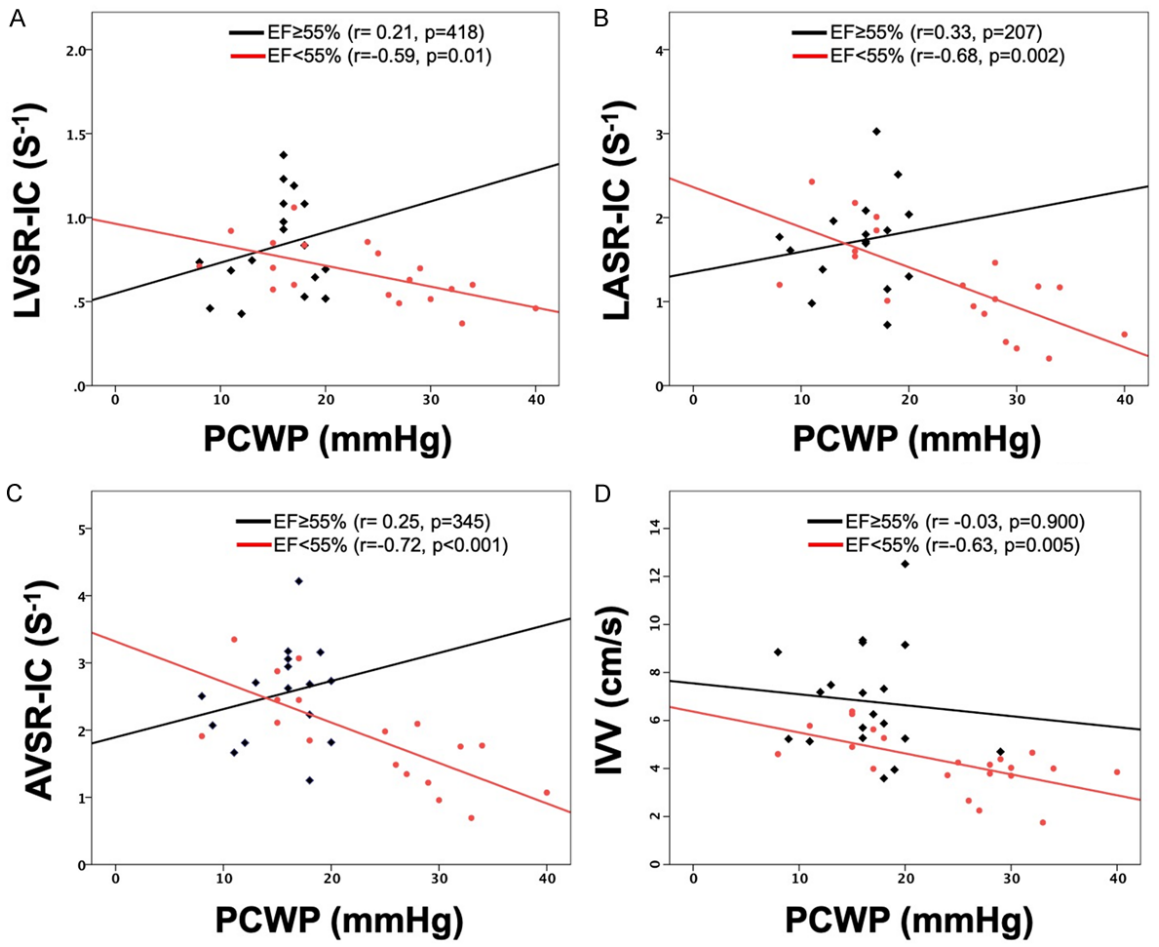

Figure 2. Correlations between different isovolumic contraction (IC) parameters with pulmonary capillary wedge pressure (PCWP). A. PCWP versus left ventricular strain rate during IC (LVSR-IC). B. PCWP versus left atrial strain rate during IC (LASR-IC). C. PCWP versus atrioventricular strain rate during IC (AVSR-IC). D. PCWP versus tissue Doppler derived mitral annular isovolumic contraction velocity (IVV). Red dots represent patients with EF≥55%, and black dots represent EF<55%.

cal, demographic, and echocardiographic data overall and in the subgroups. Briefly, there was no difference between both groups regarding age, sex, New York Heart Association (NYHA) functional class, risk factors, or medications given at the time of the study. Patients with EF<55%, however, had higher E wave velocity, lower s' velocity, larger end diastolic volume (EDV) and end systolic volume (ESV), and higher E/A, E/e' suggestive of elevated left ventricular filling pressures (LVFP), however A wave velocity, E-wave deceleration time (E-DcT), e' velocity, tricuspid regurgitation pressure gradient (TRPG), and left atrial volume were not different between both groups.

Regarding invasive pressure measurements, patients with EF<55% had higher PCWP, and pulmonary artery pressures.

Velocity and longitudinal strain rate during IC

Tissue Doppler-derived averaged mitral annular velocities during IC (IVV) was significantly lower in patients with EF<55%. On the other hand, in patients with EF<55% the average LV longitudinal strain rate tended to be lower during IC (LVSR-IC) and significantly lower during ejection (LVSR-Ej), while both LA longitudinal strain rate during IC and ejection were significantly lower when compared to patients with EF≥55%. The largest differences however were noted for atrioventricular strain rate during IC (AVSR-IC) and ejection (AVSR-Ej) both were significantly lower in patients with EF<55%.

Importantly, while E/A, E/e', LAV, IVV, LVSR-IC, LVSR-Ej, LASR-IC, LASR-Ej, AVSR-IC, and AVSR-Ej correlated with PCWP overall, all these

Table 2. Correlations with pulmonary capillary wedge pressure overall and in subgroups

	All (n=35)		EF≥55% (n=17)		EF<55% (n=18)		Multivariate Adj r^2=0.48	
	r	p	r	p	r	p	beta	p
E/A	0.50	0.003	0.25	0.332	0.45	0.08	--	--
E/e'	0.52	0.001	0.24	0.360	0.47	0.04	0.01	0.967
LAV (ml)	0.62	<0.001	0.29	0.275	0.7	0.001	0.23	0.463
IVV (cm/s)	-0.44	0.007	-0.03	0.900	-0.63	0.005	-0.15	0.67
LVSR-IC (s^{-1})	-0.39	0.029	-0.21	0.418	-0.59	0.01	-0.17	0.631
LASR-IC (s^{-1})	-0.56	0.001	-0.33	0.207	-0.68	0.002	0.48	0.631
AVSR-IC (s^{-1})	-0.58	<0.001	-0.25	0.345	-0.72	<0.001	-0.72	0.004
LVSR-Ej (s^{-1})	-0.49	0.003	-0.15	0.561	-0.55	0.02	-0.15	0.587
LASR-Ej (s^{-1})	-0.54	0.007	-0.40	0.13	-0.2	0.428	--	--
AVSR-Ej (s^{-1})	-0.53	0.001	-0.36	0.168	-0.41	0.09	--	--

LAV, left atrial volume, IVV tissue Doppler derived mitral annular velocity during isovolumic contraction, LVSR-IC, left ventricular longitudinal strain rate during isovolumic contraction, LASR-IC, left atrial longitudinal strain rate during isovolumic contraction, AVSR-IC, atrio-ventricular longitudinal strain rate during isovolumic contraction, LVSR-Ej, left ventricular longitudinal strain rate during ejection, LASR-Ej, left atrial longitudinal strain rate during ejection, AVSR-Ej, atrio-ventricular longitudinal strain rate during ejection.

Table 3. Correlations with tissue Doppler derived mitral annular velocity during isovolumic contraction (IVV) overall and in subgroups

	All (n=35)		EF≥55% (n=17)		EF<55% (n=18)		Multivariate Adj r^2=0.48	
	r	p	r	p	r	p	beta	p
LVSR-IC (s^{-1})	0.52	0.002	0.37	0.163	0.65	0.01	0.48	0.045
LASR-IC (s^{-1})	0.53	0.002	0.29	0.288	0.71	0.002	0.42	0.08
AVSR-IC (s^{-1})	0.61	<0.001	0.39	0.148	0.77	0.001	0.77	0.001

LVSR-IC, left ventricular longitudinal strain rate during isovolumic contraction, LASR-IC, left atrial longitudinal strain rate during isovolumic contraction, AVSR-IC, atrio-ventricular longitudinal strain rate during isovolumic contraction.

parameters lost correlation in patients with EF≥55% (**Figure 2**, **Table 2**), however in patients with EF<55%, E/e', LAV, IVV, LVSR-IC, LASR-IC, AVSR-IC, and LVSR-Ej negatively correlated with PCWP, while, LASR-Ej and AVSR-Ej did not (**Figure 2**, **Table 2**). Multivariate regression showed that only AVSR-IC remained to correlate with PCWP while all other parameters lost correlation (**Table 2**).

Finally, it was also noted that IVV correlated with LVSR-IC, LA-SR-IC and AVSR-IC overall, however this correlation was lost in patients with EF≥55% and remained for patients with EF<55%. Multivariate regression analysis revealed that the correlation was best in case of AVSR-IC in patients with EF<55% (**Table 3**).

Discussion

The findings of our study can be summarized as follows: first, mitral annular velocities during isovolumic contraction (IC) are decreased in patients with depressed EF compared to patients with preserved EF and in those patients, it correlates with PCWP. Longitudinal deformation derived by color tissue Doppler seems to follow a similar pattern both from the left atrium and left ventricle however when atrioventricular values were taken into consideration the lowest levels of deformation and the best correlation with PCWP were obtained in patients with depressed EF compared to either alone.

Mitral annular velocities, atrioventricular deformations, and mechanical coupling

We have shown before that tissue Doppler-derived mitral annular velocity during isovolumic contraction (IVV) correlates with PCWP in patients with depressed EF, unlike those with preserved EF [4]. The mechanism of IVV correlation with PCWP seems to be based on the deformation that occurs during IC and ejection, respectively. Changes in LV and LA mechanics

within the normal conical heart are reported to be load-dependent [7]. Conversely, dilated and failing hearts exhibit different mechanical responses, independent of loading conditions, because cardiac fiber orientation is responsible for such changes [4]. Accordingly, the lack of correlation with PCWP in patients with preserved EF noticed in our study as well as our previous studies seems reasonable.

However, it is to be noted that, most of the mitral annulus is a fibrous structure that does not exhibit deformation [10]. As such, mechanical changes observed at the level of the mitral annulus may just be a result of tethering to the walls of the LV and LA. All previous studies of relationships of mitral annular velocities assume a closer indirect relationship to the LV deformational changes, however, our hypothesis suggests that mitral annular mechanical behavior may actually be a representation of atrioventricular (AV) mechanical coupling. AV mechanical coupling refers to the dependence of better cardiac function on the co-existing functions of the LA and the LV despite structural and functional differences [11]. Examining the LV and LA muscle fibers and mechanical behaviors yields considerable differences. While the LV muscle is thicker than the LV and LV fibers are arranged in helices to produce deformations in longitudinal, radial, circumferential, and rotational directions, the LA fibers are predominantly longitudinal to produce functions in three successive phases namely reservoir, conduit, and booster. Recently, it was shown that AV coupling is essential for normal cardiac function such that atrial dysfunction may indicate diastolic heart failure [12], however, coupled increased ventricular systolic deformation potentially compensates for diastolic properties. As such, in patients with normal EF, none of these variables will correlate with filling pressures, unlike patients with depressed EF. This may explain our observations that the attenuation of both LVSR-IC and LASR-IC and the correlation with PCWP is almost identical in patients with EF<55% with a lack of such correlation in patients with EF≥55%. Combining both longitudinal deformations, however, yielded the strongest associations with parameters of systolic and diastolic dysfunction, pointing towards the role of AV mechanical coupling in these patients.

Combined LA-LV deformational imaging: reported value that extends into isovolumic contraction

The additive effect of combining the LA and LV longitudinal strains in the assessment of myocardial dysfunction has previously been reported. Cameli et al. reported that studying LA and LV in isolation is inappropriate, given that they represent a true single morpho-functional unit [13]. As such, they have tested the combined AV longitudinal strain as the sum of their absolute peak values in two- and four-chambers views during the same cardiac cycle in patients with cardiac risk factors and showed that this is a useful tool to identify subclinical myocardial dysfunction. In another study by Addetia et al. similar concept was tackled by simultaneous measurement of LS in all cardiac chambers to study inter-chamber functional relationships in different cardiac disease states. In our study, we have used strain rate rather than strain because the hypothesis focuses on the deformation during isovolumic contraction [14]. Despite that, our findings go in a similar direction with the aforementioned studies, especially that relationships of deformations during ejection were reproduced in our study. Our study however extends these relationships to parts of the cardiac cycle where deformation is not usually assessed. Our study suggests that AV coupling starts in the isovolumic contraction, a finding that will shed further light on the pathophysiology of myocardial dysfunction and heart failure and may help further phenotypic characterization of patients in health and in disease.

Several limitations should be considered. First, this is a small pilot study and larger studies with a control group should be done to confirm our findings. Second, color tissue Doppler is limited by angle dependency and low signal-to-noise ratio, however, it was the method of choice for us considering the high frame rate suitable for capturing velocities occurring at fast occurring phases such as IC. Third, to study the true effect of atrioventricular mechanical deformation during various phases of the cardiac cycle, simultaneous measurement should be obtained, further studies should test the reproducibility of the findings reported here when values are measured simultaneously from the LA and the LV. Finally, assessment of car-

diac deformation in other directions during IC should be assessed to confirm the study findings.

Conclusions

Combined LA and LV strain rate during isovolumic contraction is a powerful predictor of left ventricular filling pressures in patients with depressed EF and not in patients with preserved EF and can be explained by the concept of mechanical coupling where hemodynamically significant cardiac dysfunction occurs when LV deformations fail to compensate for LA dysfunction. The correlation previously mentioned between mitral annular velocities and PCWP in the same group of patients can be related to the tethering of the fibrous structure to both chambers and as a result, may indicate that mitral annular velocities are an indirect representation of AV mechanical coupling. Importantly, while AV mechanical coupling was previously reported to be essential for normal cardiac function, our study is the first to report that such important mechanical behavior starts prior to ejection, a finding that can be useful in understanding and stratification of heart failure patients in the future.

Disclosure of conflict of interest

None.

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