Left Ventricular Diastolic Filling Patterns as Predictors of Heart Failure After Myocardial Infarction: A Colour M-Mode Doppler Study

Elena Kinova, Hristo Kozhuharov

Department of Cardiology, University Hospital Queen Joanna, Sofia, Bulgaria

Key words:
Diastolic function, echocardiography, flow propagation velocity.

Introduction: Combined assessment of mitral filling pattern and colour M-mode flow propagation velocity (Vp) allows separation of the effects of compliance and relaxation on left ventricular filling, thereby allowing identification of pseudonormal filling, which may be valuable after myocardial infarction. We sought to investigate prognostic implications of left ventricular filling patterns, determined by mitral E- and A-wave velocities and Vp, in relation to development of heart failure after myocardial infarction.

Methods: Echocardiography was performed in 91 consecutive patients within 72 hours after myocardial infarction and at day 15. Patients were divided into: Group 1 – with normal filling (E-wave /A-wave ratio ≥1 and E-wave /Vp ratio <1.5) and impaired relaxation (E/A <1 and E/Vp <1.5 or ≥1.5) and Group 2 – with pseudonormal and restrictive filling (E /A ≥1 or 2 and E /Vp ≥1.5). The period of follow-up was 5.5 months.

Results: There were 56 patients in Group 1 and 35 patients in Group 2, respectively. Patients from Group 2 were in significantly worst Killip class than patients in Group 1 ($p<0.0001$, Mann-Whitney U test). The combination of E/A and E/Vp ratios proved to be superior to single parameters of left ventricular function in relation to in-hospital heart failure ($p<0.0001$, test-value 0.69). Ten patients were readmitted for heart failure during the 5.5 months of follow-up. Cox analysis identified E-wave deceleration time <140 ms and combination of E/A ratio ≥1 or 2 and E/Vp ratio ≥1.5 to be independent predictors of worsening of heart failure.

Conclusion: The combination of E/A ratio ≥1 or 2 and E/Vp ratio ≥1.5, that defined pseudonormal or restrictive filling, correlates with in-hospital heart failure after first myocardial infarction and is the second predictor of worsening heart failure after deceleration time less than 140 ms.

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Corresponding Author:
Elena Kinova

Studentski grad bl. 58
entr. A apt. 702
1734 Sofia, Bulgaria
e-mail:
ekinova@hotmail.com

The importance of assessment of diastolic function is emphasized by the fact that Doppler echocardiographic measurements have been shown to be significantly related to functional status[1] and contain prognostic information independent of left ventricular systolic function in patients with heart failure[2].

In recent years, Doppler echocardiography has been accepted as a noninvasive alternative to cardiac catheterization for assessment of left ventricular diastolic function[3]. The flow propagation velocity of early transmitral flow (Vp), measured by colour M-mode Doppler, has been proposed as a new, relatively preload-independent, index of left ventricular relaxation[4-7] which allows noninvasive determination of left ventricular filling pressures[8] and identification of a pseudonormal mitral filling pattern. Therefore, combined assessment of Doppler mitral flow measurements and flow propagation velocity Vp may be used for identifi cation of patients in high risk for adverse events.

The aim of this study was to investigate prognostic implications of left ventricular filling patterns, as defined by mitral peak E- and A-wave velocities and colour M-mode flow propagation velocity, in relation to development of heart failure after first acute myocardial infarction.

Methods

Study population

We prospectively studied 91 consecutive patients who were admitted to the Clinic of Cardiology at Queen Joanna Hospital, with first myocardial infarction diagnosed by at least two of the following criteria: 1. Characteristic chest pain, 2. Electrocardiographic signs of acute myocardial infarction, 3. Transient elevation of creatinine kinase >210 IU/L (at least twice the upper limit). Patients with previous myocardial infarctions, atrial fibrillation, left bundle branch block, significant heart valve diseases and cardiomyopathies were not included. Data on medical history, physical examination, laboratory results, electrocardiograms and medication treatment were prospectively collected for all patients. Follow-up data was collected at a median interval of 5.5 months (range 5 to 6 months) when patients were examined. Readmissions to hospital for heart failure, revascularizing procedures or death, were noted. The local Ethics Committee approved the study and each participant gave informed written consent.

Echocardiography

Echocardiography was performed on a Hewlett Packard (Sonos 2500) with a 2.5 MHz transducer, within 72 hours of myocardial infarction and on day 15. Images were stored on a videotape recorder for further analysis independently of the clinical data. For each Doppler variable, five consecutive beats were measured and averaged.

Pulsed Doppler echocardiography

Mitral flow velocity was obtained with Doppler beam perpendicular to the plane of the mitral annulus and sample volume between the tips of mitral leaflets in the apical four chamber view. The left ventricular isovelocity relaxation time (IVRT) was measured from Doppler recordings, in the apical five-chamber view with the sample volume between the left ventricle outflow tract and the mitral valve, that show both aortic valve closure and mitral flow[3].

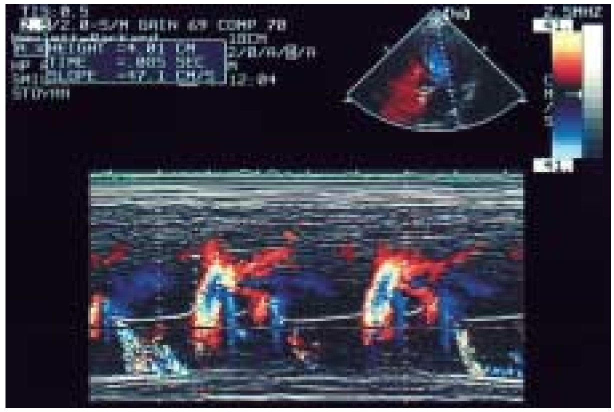

Figure 1. The flow propagation velocity (Vp) was measured as the slope of the first aliasing velocity from the mitral annulus in early diastole to 4 cm into the left ventricular cavity.

Colour M-mode Doppler echocardiography

Colour M-mode Doppler echocardiography was carried out in the apical four chamber view with the cursor aligned parallel to left ventricular inflow and positioned through the center of the flow, avoiding boundary regions. The flow propagation velocity was measured as the slope of the first colour aliasing velocity from the mitral annulus in early diastole to 4 cm distally into the cavity (Figure 1). If patients had low mitral E-wave velocity and no aliasing was seen, baseline shift was adjusted to alias at about 75% of the mitral E-wave velocity.

Diastolic filling pattern was classified as: normal – peak E-wave/peak A-wave ratio (E/A) ≥1 and peak E-wave/flow propagation velocity ratio (E/Vp) <1.5; impaired relaxation – E/A <1 and E/Vp <1.5 or ≥1.5; pseudonormal filling – E/A ≥1-2 and E/Vp ≥1.5; restrictive filling – E/A ≥2 and E/Vp ≥1.5.

Patients were divided into: Group 1 – with normal filling and impaired relaxation and Group 2 – with pseudonormal and restrictive filling (E-wave/A-wave ratio ≥1 or 2 and E-wave/Vp ratio ≥1.5). The presumption was that the patients in Group 1 had no high compensatory preload and that in Group 2 there was increased preload compensation.

Evaluation of left ventricular systolic function

Left ventricular volumes and ejection fraction were calculated from the apical four-chamber and two-chamber views using the apical biplane method of discs. The wall motion score index was obtained semiquantitatively using a 16-segment model[9].

Table 1. Baseline demographic and clinical variables.

	Group 1 n=4+52	Group 2 n=29+6	p-value
Men / women	28/28 (50%)	20/15 (57%)	NS
Age	65±11	59±13	0.02
History of hypertension	45 (80%)	24 (69%)	NS
Smoking	27 (48%)	17 (49%)	NS
Q-wave myocardial infarction	34 (61%)	26 (74%)	NS
Anterior myocardial infarction	36 (64%)	12 (34%)	NS
Use of thrombolytic therapy	10 (18%)	12 (34%)	NS
Peak creatine kinase (IU/L)	1092±1125	1517±1464	NS
Use of diuretics	17 (30%)	19 (54%)	0.02

Data are expressed as the number (%) of patients or mean value ± standard deviation, p-value = comparison between the two groups, NS = non-significant.

Flow mapping of the mitral regurgitant jet, if presented, was performed with colour flow Doppler in both an apical four- and two-chamber view. Based on this evaluation, regurgitation is graded on a 0 to 4+ scale, where 1+ is mild, 2 to 3+ is moderate and 4+ is severe regurgitation[10].

One operator performed all echocardiographic studies and intraobserver variability was under 10% for all Doppler parameters in fifteen randomly chosen patients.

The degree of heart failure, expressed as the worst Killip class during hospitalization, was determined by cardiologists who were unaware of the echocardiographic findings. Heart failure was defined according to Killip and Kimball[11]: class I - no presence of clinical signs of left ventricle dysfunction, class II – with presence of a S3 gallop and/or mild/moderate pulmonary congestion (<50% of the lungs), class III –pulmonary oedema (> 50% of the lungs) and class IV – cardiogenic shock.

Patients were treated with following medications: streptokinase 24%, aspirin 80%, heparin 94%, nitrates 97%, b-blockers 49%, angiotensin-converting enzyme inhibitor 49%, diuretics 40%.

Statistics

Continuous data are presented as mean ± standard deviation. Comparison between groups was performed using independent-samples t-test and Mann-Whitney U-test for continuous parametric and non-parametric variables, respectively. Categorical data were compared using chi-square and Gamma test, where appropriate. Paired-sample t-tests were used for in-group comparison. Multivariate Cox proportional hazards analyses were performed to identify independent predictors of readmission for heart failure and cardiac death. Variables included in the multivariate analyses were selected by univariate analyses. Values of $p < 0.05$ were considered to be significant. Statistical analysis was performed using SPSS (Chicago, Illinois) -version 10.0 for Windows.

Results

The average age of the patients was 62±12 years, range 28 to 93 years (53% men, 47% women). There were 56 patients in Group 1 (4 with normal filling and 52 with impaired relaxation) and 35 patients in Group 2 (29 with pseudonormal filling and 6 with restrictive filling). Demographic and clinical variables are summarized in Table 1. Statistically significant differences in age and use of diuretics were found between the two groups - in Group 2 the mean age was lower ($p=0.02$) and the use of diuretics was more frequent ($p=0.02$).

Echocardiographic evaluation of left ventricular function

The echocardiographic data are summarized in Table 2. Between the two groups there was statistically significant difference ($p<0.0001$) in all mitral flow indices and E/Vp ratio with higher E/A and E/Vp ratios in Group 2.

In Group 1 at day 15 the mean values of E-wave velocity ($p=0.004$), E-wave/A-wave ratio ($p=0.05$), E-wave/Vp ratio ($p=0.002$) and end-diastolic volume

Table 2. Echocardiographic variables at admission.

	Group 1 n=4+52	Group 2 n=29+6	p-value
Peak E-wave velocity (cm/s)	60.8±15.4	89.8±22.4	<0.0001
Peak A-wave velocity (cm/s)	87.8±23.0	62.5±17.6	<0.0001
E/ratio	0.7±0.2	1.4±0.7	<0.0001
E-wave deceleration time (ms)	222.0±50.1	170.6±34.8	<0.0001
Flow propagation velocity V (cm/s)	39.5±10.5	44.6±11.4	0.03
E/Vp ratio	1.5±0.5	2.1±0.5	<0.0001
Isovolumic relaxation time (ms)	114.5±16.4	93.0±23.0	<0.0001
End-diastolic volume index (ml/m^2)	45.7±12.5	56.6±17.7	0.001
End-systolic volume index (ml/m^2)	23.4±8.8	30.9±12.5	0.001
Ejection fraction (%)	49.6±8.7	46.4±7.7	NS
Wall motion index	1.7±0.3	1.9±0.4	0.04
Mitral regurgitation ≥2+	12 (21%)	16 (45%)	0.02

Data are expressed as the mean value ± standard deviation or number (%) of patients, p-value = comparison between the two groups, NS = non-significant.

index (p=0.02) were significantly increased and A-wave velocity decreased (p=0.01). The flow propagation velocity was not significantly changed (p=0.2). In Group 2, A-wave velocity (p=0.04) and E-wave deceleration time (p=0.02) were significantly increased. The other echocardiographic parameters at day 15 did not significantly differ. The patient distribution according to diastolic filling pattern was: 3 patients with normal filling, 53 patients with abnormal relaxation, 25 patients with pseudonormal filling and 6 with restrictive filling. In one patient percutaneous transluminal angioplasty was performed and he was excluded from the analysis. Two patients with initially restrictive filling and one with impaired relaxation died within first week of the infarction.

Heart failure

Development of in-hospital heart failure was not observed in patients with normal left ventricular diastolic filling. Forty-one patients (45%) had signs of in-hospital heart failure. The distribution of the Killip class in the two groups of diastolic function was: Group 1 - 39 patients in Killip class I and 17 patients in Killip class II, Group 2 – 11 patients in Killip class I, 19 patients in Killip class II, 3 patients in Killip class III and 2 patients in Killip class IV. Patients with pseudonormal and restrictive filling (Group 2) were in significantly higher Killip class than patients in Group 1 (p<0.0001, Mann-Whitney U test). Three patients from all with in-hospital heart failure had significant systolic dysfunction, represented by an ejection fraction <40% and impaired relaxation, 4 patients had significant systolic dysfunction and pseudonormal / restrictive diastolic filling, 20 patients had pseudonormal / restrictive filling but without significant systolic dysfunction and 14 had impaired relaxation and ejection fraction ≥40% (Figure 2).

Significance, strength and direction of relationship between clinical and echocardiographic va-

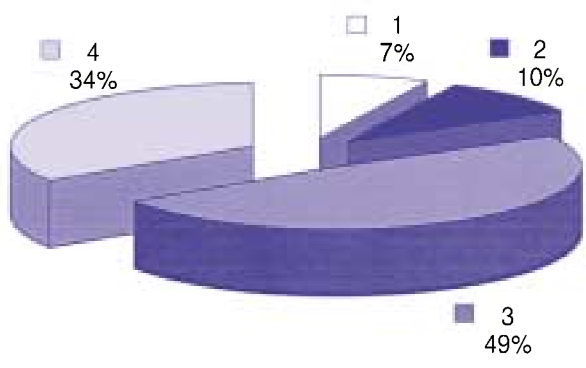

Figure 2. Distribution of impairment of left ventricular systolic and diastolic function in patients with in-hospital heart failure.
1 - Significant systolic dysfunction and impaired relaxation
2 - Significant systolic dysfunction and pseudonormal/restrictive filling
3 - Pseudonormal/restrictive filling without significant systolic dysfunction
4 - Impaired relaxation without significant systolic dysfunction

Table 3. Significance, strength and direction of relationship between clinical and echcardiographic variables at admission and in-hospital heart failure.

	Gamma test-value	p
E/A ≥1 or 2 and E/Vp ≥1.5	0.69	<0.0001
Peak creatine kinase >300 IU/L	0.67	<0.0001
E/Vp ratio ≥1.5	0.57	0.004
Q-wave myocardial infarction	0.52	0.005
Wall motion index >1.7	0.50	0.005
E/A ratio >2	0.93	0.01
Mitral regurgitation ≥2+	0.46	0.02
Ejection fraction <40%	0.54	NS
Isovolumic relaxation time <70 ms	0.53	NS
E-wave deceleration time <140 ms	0.48	NS
Use of thrombolytic therapy	0.34	NS
Flow propagation velocity <45 cm/s	0.19	NS
Age >65 years	-0.15	NS

E/A = ratio of peak E-wave velocity to peak A-wave velocity, E/Vp = ratio of peak E-wave velocity to flow propagation velocity, NS = non-significant.

riables and in-hospital heart failure was tested with a Gamma test (Table 3). The combination of E-wave/A-wave ratio and E-wave/Vp ratio correlated to Killip class (p<0.0001, test-value 0.69).

Clinical status at follow-up

Hospital readmission for heart failure during 5.5 months of follow-up occurred in 10 patients (11%). Two of them were from Group 1 (4%) and 8 from Group 2 (20%), p=0.005. Most of them (9 patients) had in-hospital heart failure (p=0.005), 7 had a Q-wave myocardial infarction (p=0.01) and thrombolytic therapy was performed in only 3 of them (NS). These patients had higher E-wave / A-wave ratio (p<0.0001) and E-wave / Vp ratio (p=0.003) and shorter deceleration time (p=0.001) and isovolumic relaxation time (p=0.01) at admission comparing to patients without worsening of heart failure. There was no significant difference in ejection fraction and wall motion index. Multivariate Cox proportional hazards analysis were performed to identify independent predictors of readmission for heart failure (Table 4). Deceleration time <140 ms (Odds Ratio [95%CI] = 6.1 [1.7-21.8], p=0.005) and pseudonormal / restrictive filling, as determined by E/A ≥1 or 2 and E/Vp ≥1.5 [Odds Ratio [95%CI] =5.5 [1.2-25.9], p=0.03) proved to be predictors of worsening of heart failure.

Table 4. Multivariate predictors of hospital readmission for heart failure and cardiac death.

	B	Wald	Relative risk (95% CI)	p
Hospital readmission for heart failure				
Deceleration time <140 ms	1.9	7.9	6.1 (1.7 – 21.8)	0.005
E/A ≥1 or 2 and E/Vp ≥1.5	1.7	4.6	5.5 (1.2 – 25.9)	0.03
Cardiac death				
Isovolumic relaxation time <70 ms	2.9	18.0	18.7 (4.8 – 72.3)	< 0.0001
Ejection fraction <40%	2.0	8.3	7.3 (1.9 – 28.5)	0.004
Age >65 years	2.0	5.7	7.4 (1.4 – 38.1)	0.002

B=coefficient; Wald=$(B/S.E)^2$

Revascularization procedures were performed in 3 of the patients who were not included in the analysis.

Cardiac death occurred in 11 patients (12%), 7 of them were with initially impaired relaxation, 1 with pseudonormal diastolic pattern and 3 with restrictive diastolic filling. Three patients died within the first week of myocardial infarction: one from cardiac rupture and two from heart failure. Eight patients died during the out-of hospital period: 3 patients from sudden cardiac death and 5 patients from heart failure (3 of them had second infarctions). The mean age of patients with cardiac death was 68±8 vs. 62±12 years in patients who survived ($p=0.04$). Three patients were in Killip class I during hospitalization and 8 had in-hospital heart failure. These patients had higher E-wave/A-wave ratio ($p=0.03$) and E-wave/Vp ratio ($p=0.04$) and shorter isovolumic relaxation time ($p=0.05$) at admission compared to survivors. As regards to ejection fraction and wall motion index, no statistically significant difference was found. Multivariate Cox proportional analysis was performed (Table 4) and isovolumic relaxation time <70 ms was the best predictor of cardiac death (Odds Ratio [95%CI] = 18.7 [4.8-72.3], $p < 0.0001$).

Discussion

Left ventricular diastolic filling pattern

Acute myocardial ischemia is associated with a marked change in the early diastolic intraventricular flow pattern, which is delayed in the apical region[12]. In patients with relatively small infarcts a pattern of abnormal relaxation is the most frequent finding[13]. In our study these patients represent 57% of the study population and ejection fraction was >40%.

As abnormal relaxation and increased chamber stiffness have opposing effects on left ventricular filling, when both abnormalities are present at the same time, an elevated compensatory left atrial pressure normalizes the early diastolic transmitral pressure gradient and velocity, despite impaired left ventricular relaxation[14]. Consequently, the ability of the pulsed Doppler recording to identify pseudonormal mitral flow velocity profile may be significantly compromised. Analysis of pulmonary venous flow variables though helpful, is technically challenging and is also affected by rhythm, heart rate and respiration[15].

Whereas pulsed wave Doppler echocardiography allows determination of time and velocity at a single location, the colour M-mode technique has the ability to visualize the propagation of flow along the entire length of the left ventricle throughout diastole, thereby allowing analysis of time, velocity and space[5]. Simultaneous hemodynamic and Doppler echocardiographic studies have indicated that flow propagation velocity Vp could represent a noninvasive index for assessment of LV relaxation[4,5,12] and is a relatively preload-independent index of left ventricular filling[6]. Reduced flow propagation velocity Vp has been shown to be present during myocardial ischemia[12] as well as in the acute phase of myocardial infarction[16] and to be decreased in patients with normal E/A ratio but elevated filling pressures, indicating this technique to be useful in detecting pseudonormal left ventricular mitral filling patterns[4,17]. In this study, 32% of patients were classified as having pseudonormal filling and 62% of them had symptoms of in-hospital heart failure.

On the other hand, left ventricular flow propagation velocity was studied before and after hemodialysis with ultrafiltration in hemodialysis patients[18]. It is concluded that pseudonormalization, which was due to volume overload before hemodialysis, resulted in underestimation of the degree of diastolic dysfunction. Therefore, the advantage of colour M-mode flow propagation velocity as a single parameter over conventional parameters for the assessment of LV diastolic function is limited. This may explain higher Vp >45 cm/s in some patients in Group 2. Flow propagation velocity was influenced by preload compensation and was the reason Vp was combined with with mitral E-wave.

A restrictive filling pattern is more common in patients with greater myocardial infarction and identifies a subgroup at particularly high risk of an adverse outcome in patients with depressed systolic function[19-21]. Previous investigations have reported a relatively high prevalence of restrictive filling in early phase of myocardial infarction – 13% in the study of Nijland[19], 14% in that of Sakata[20] and 19% in that of Burgess[22]. In this study 7% of patients were with initially restrictive filling but we considered as restrictive filling pattern only mitral flow pattern with E/A ≥2 and not with E/A ratio 1-2 and deceleration time <140 ms[22]. Another reason for the small number of patients was that patients who died within 24-48 hours of the event were not included. Nevertheless, the event rate in patients with restrictive filling pattern was high.

Heart failure and survival

Poulsen et al[23] have reported 36% of patients have had in-hospital congestive heart failure, while a higher frequency was found by Moller et al – 48%[17]. In this study 45% of patients had symptoms of heart failure. Several previous studies have shown good correlation between symptoms of heart failure and diastolic dysfunction[1,24-26]. In the acute phase of myocardial infarction, restrictive filling has shown to be predictive of development of heart failure due to marked elevation of left ventricular end-diastolic pressure[21,23,26,28]. However, E/Vp ≥1.5 is an even better and more sensitive predictor as it is suggestive of moderately elevated left ventricular end-diastolic pressure. Ueno et al[27] concluded that E/Vp by colour M-mode Doppler provides a better estimate of pulmonary capillary wedge pressure than transmitral or pulmonary venous flow in patients with acute myocardial infarction. In the current study 59% of patients with in-hospital heart failure had pseudonormal / restrictive filling and only 4 of them had an ejection fraction <40%. Moreover, the ejection fraction was not significantly different between patients with and without readmission for heart failure that is a confirmation that left ventricular diastolic parameters are superior to systolic parameters, in relation to heart failure.

We found higher prevalence of anterior myocardial infarction in patients from Group 1 but 61% of them were non-Q-wave myocardial infarctions. This was an explanation of lower rate of thrombolytic treatment, lower wall motion index and rare moderate to severe mitral regurgitation in Group 1.

Several studies[19,23,28] have shown that advanced diastolic dysfunction with decreased duration of deceleration time is a powerful predictor of heart failure or cardiac death after myocardial infarction. In the current study deceleration time <140 ms was found to be a predictor of readmission for worsening of heart failure in Cox analysis but the duration of isovolumic relaxation time <70 ms was the best predictor of cardiac death. Müller at al[17,29] have reported the ratio of E-wave velocity to flow propagation velocity E/Vp to be a strong predictor of heart failure and death. Our results indicated that there was relationship between E/Vp ratio and in-hospital heart failure but it was weaker than that of peak creatinine kinase. However, the combination of E/A ≥1 or 2 and E/Vp ≥1.5 that defined pseudonormal and restrictive filling, correlated most with in-hospital heart failure and proved to be the second predictor of readmission for heart failure. The ratio E/Vp ≥1.5 is useful, in addition to mitral Doppler indices, in identifying patients with elevated left ventricular diastolic pressure but not as a single parameter because of the risk of misclassifying patients with impaired relaxation and markedly reduced flow propagation velocity in high risk Group. Thirty-one patients from Group 1 had E/A < 1 and E/Vp ≥1.5. However, the ratio E/Vp may add important information if E/A is ≥1 with identifying pseudonormal diastolic filling.

Study limitations

The study population was relatively small and did not include patients with cardiac death in the first 24-48 hours. We lost a relatively large number of patients with probably restrictive / pseudonormal filling pattern, however, our aim was to find the predictors of heart failure in survivors. CPK-MB was not measured because of technical reasons and this may influence diagnosis in same cases. However, we did not include patients suffering from other diseases that may increase CPK in our study. Thrombolytic therapy was used in 37% of patients with Q-wave myocardial infarction and the use of revascularization procedures was low, possibly resulting in higher frequency of heart failure. But this is the reason for the great importance of prognostic implications of left ventricular echocardiographic parameters in relation to development of heart failure in our patient population. In this study no invasive hemodynamic procedures were performed. However, the parameters of Doppler mitral flow[30,31] and colour M-mode Doppler[7] have previously been investigated with simultaneous invasive measurements that are the scientific base of our study. The criteria of pseudonormal (E/A ≥1-2 and E/Vp ≥1.5) and restrictive filling pattern (E/A ≥2 and E/Vp ≥1.5) have not been established with hemodynamic measurements. Using the combination of E/A and E/Vp ratios for classification of diastolic function may lead to some mistakes – patients with pseudonormal filling pattern may actually have restrictive filling pattern or some patients classified as with normal diastolic function may have pseudonormal filling pattern. However, our hypothesis that the combination of E/A ≥1 or 2 and E/Vp ≥1.5 identifies pseudonormal / restrictive diastolic filling pattern was fully supported by the established correlation with in-hospital heart failure and prediction of worsening of heart failure. This needs further evaluation with invasive hemodynamic methods. Although we used prone to subjectivity tracing method to measure the slope of flow propagation velocity, instead of more accurate method with special computer software[32], the intraobserver variability was within admissible limits.

Conclusion

The combination of E/A ratio ≥1 or 2 and E/Vp ratio ≥1.5, that defined pseudonormal or restrictive filling, correlates most with in-hospital heart failure after the first myocardial infarction and is the second predictor of worsening of heart failure after deceleration time less than 140 ms.

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