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Noninvasive Estimation of Left Ventricular Filling Pressure by E/e′ Is a Powerful Predictor of Survival After Acute Myocardial Infarction

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OBJECTIVES	The aim of this study was to assess the prognostic value of a noninvasive measure of left ventricular diastolic pressure (LVDP) early after acute myocardial infarction (MI).
BACKGROUND	The early diastolic velocity of the mitral valve annulus (e′) reflects the rate of myocardial relaxation. When combined with measurement of the early transmitral flow velocity (E), the resultant ratio (E/e′) correlates well with mean LVDP. In particular, an E/e′ ratio >15 is an excellent predictor of an elevated mean LVDP. We hypothesized that an E/e′ ratio >15 would predict poorer survival after acute MI.
METHODS	Echocardiograms were obtained in 250 unselected patients 1.6 days after admission for MI. Patients were followed for a median of 13 months. The end point was all-cause mortality.
RESULTS	Seventy-three patients (29%) had an E/e′ >15. This was associated with excess mortality (log-rank statistic 21.3, p < 0.0001) and was the most powerful independent predictor of survival (risk ratio 4.8, 95% confidence interval 2.1 to 10.8, p = 0.0002). The addition of E/e′ >15 improved the prognostic utility of a model containing clinical variables and conventional echocardiographic indexes of left ventricular systolic and diastolic function (p = 0.001).
CONCLUSIONS	E/e′ is a powerful predictor of survival after acute MI. An E/e′ ratio >15 is superior, in this respect, to other clinical or echocardiographic features. Furthermore, it provides prognostic information incremental to these parameters. (J Am Coll Cardiol 2004;43:360–7) © 2004 by the American College of Cardiology Foundation

Advanced left ventricular (LV) diastolic dysfunction predicts a poorer prognosis after acute myocardial infarction (MI) (1–3). In particular, echocardiographic indexes of elevated LV filling pressures are associated with adverse remodeling, an increased incidence of heart failure, and worse survival (1–4). The deceleration time (DT) of early transmitral flow is the most extensively investigated echocardiographic diastolic parameter and appears to be the simplest and most powerful predictor of outcome (2–6). However, the correlation between DT and LV filling pressure (or pulmonary capillary wedge pressure [PCWP]) is relatively poor in patients with preserved systolic function (7).

To more accurately identify patients with elevated LV filling pressures, transmitral flow patterns and DT have been combined with other indicators of diastolic function (7–12). Of these, Doppler tissue imaging of mitral annulus motion appears to be particularly useful (7,12). Mitral annulus velocity reflects the rate of change in LV long-axis dimension and volume. This, in turn, is related to myocar-dial relaxation, so that impaired relaxation results in a reduced early mitral annulus velocity (e′). Unlike other Doppler parameters of diastolic function, e′ velocity appears to be relatively independent of preload, especially when the rate of myocardial relaxation is decreased (13). In addition, the ratio of early transmitral flow velocity (E) to early diastolic septal mitral annulus velocity (E/e′) (Fig. 1) has recently been shown to be the most accurate noninvasive predictor of elevated LV filling pressure (7). Therefore, we hypothesized that elevated E/e′ would predict an adverse outcome after acute MI. To test this hypothesis, we evaluated the prognostic value of E/e′ in a large series of unselected patients with acute MI and compared this with other established clinical, systolic, and diastolic parameters.

METHODS

Patients. Between August 1999 and July 2001, 593 patients were treated for acute MI at St. Mary's Hospital, Rochester, Minnesota, and also had a clinically indicated transthoracic echocardiogram during their index admission. The study group consisted of all 250 patients from this population whose echocardiographic examination included both Doppler assessment of transmitral flow velocities and Doppler tissue imaging of the medial mitral valve annulus. Myocardial infarction was defined using the European

Society of Cardiology/American College of Cardiology guidelines (14). The study was approved by the Institutional Review Board of the Mayo Clinic.

Echocardiography. Echocardiography was performed by experienced sonographers and reported by staff cardiologists with advanced training in echocardiography. All echocardiographic data used in the current study were recorded by the reviewing cardiologist at the time the scan was performed. Left ventricular systolic function was assessed semi-quantitatively using a visually estimated ejection fraction (EF) and wall motion score index (WMSI), calculated using a standard 16-segment model and five-point scoring system (1 = normal) (15). Previous work at our institution

has validated both of these methods (7,16,17). Mitral regurgitation was graded using color flow imaging, including the proximal isovelocity surface area method in 23 cases (9%) (18).

Mitral inflow was assessed in the apical four-chamber view, using pulsed-wave Doppler echocardiography, with the Doppler beam aligned parallel to the direction of flow and the sample volume at the leaflet tips. From the mitral inflow profile, the E- and A-wave peak velocities and DT were measured (19). Patients were categorized on the basis of their DT (2–6). In these analyses, DT ≤140 ms was considered abnormally abbreviated (1–3,5).

Doppler tissue imaging of the mitral annulus was obtained from the apical four-chamber view, using a 1- to 2-mm sample volume placed in the septal mitral valve annulus. Previous data have confirmed the excellent reproducibility in this measurement and have also demonstrated an E/e′ ratio >15 to be the best Doppler predictor of an elevated (>12 mm Hg) mean left ventricular diastolic pressure (mLVDP) (7). An increased E/e′ ratio was therefore prospectively defined as >15.

Baseline clinical data and patient follow-up. Baseline clinical data were obtained by chart review. Follow-up was performed between January and April 2002 by using mailed questionnaires, telephone interviews, a review of medical records, and the Social Security Agency Death Index. The study end point was death from all causes.
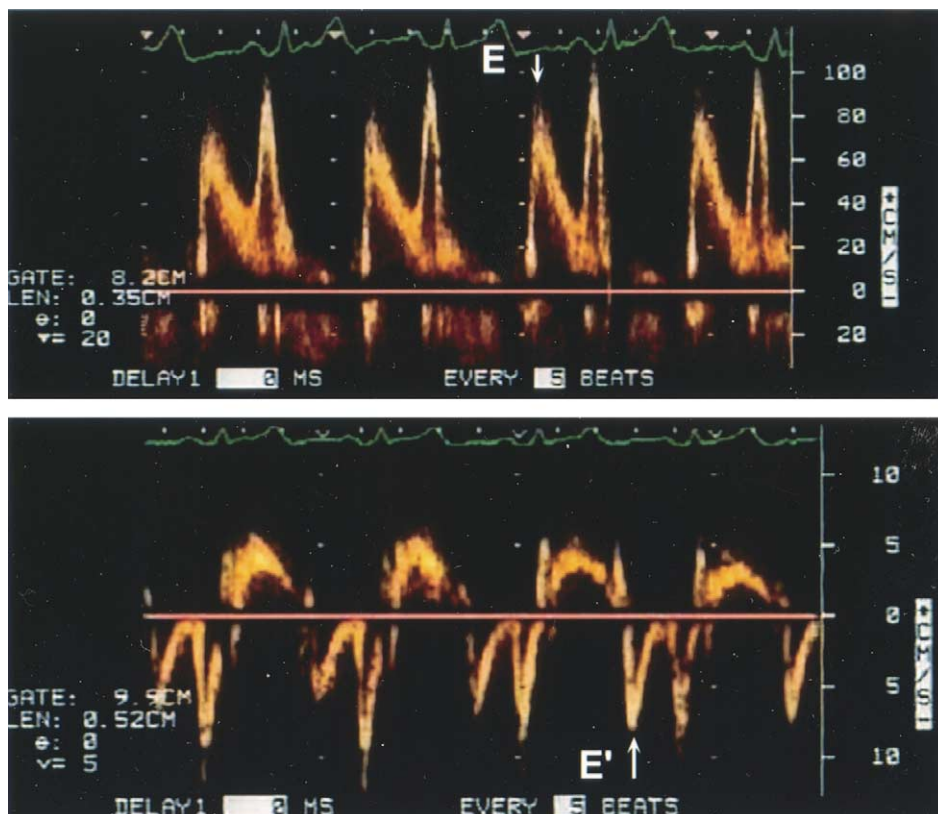

Figure 1. Transmitral Doppler **(top)** and tissue Doppler **(bottom)** imaging of the mitral annulus. The mitral E-wave velocity is 80 cm/s, and the early mitral annulus e′ velocity is 8 cm/s, giving an E/e′ ratio of 10.

Table 1. Clinical Characteristics

Characteristic	All Patients (n = 250)	E/e′ ≤15 (n = 177)	E/e′ >15 (n = 73)	p Value
Age (yrs)	68 ± 13	65 ± 13	75 ± 10	<0.001
Risk factors and medical history				
Male gender	155 (62%)	120 (68%)	35 (48%)	0.004
Current smoker	57 (23%)	44 (25%)	13 (18%)	0.25
Diabetes	56 (22%)	30 (17%)	26 (36%)	0.002
Hypertension	142 (57%)	95 (54%)	47 (64%)	0.13
Hyperlipidemia	104 (42%)	70 (40%)	34 (47%)	0.33
Previous acute MI	50 (20%)	26 (15%)	24 (33%)	0.002
Previous revascularization	44 (18%)	27 (15%)	17 (23%)	0.15
Anterior MI	106 (42%)	77 (44%)	29 (40%)	0.67
ST-segment elevation MI	120 (48%)	90 (51%)	30 (41%)	0.71
Thrombolytic therapy	39 (16%)	31 (18%)	8 (11%)	0.25
In-hospital revascularization				
PCI	160 (64%)	119 (67%)	41 (56%)	0.11
CABG	18 (7%)	9 (5%)	9 (12%)	0.06
Multivessel coronary disease*	117 (54%)	79 (50%)	38 (64%)	0.07
In-hospital therapy				
Heparin	237 (95%)	170 (96%)	67 (92%)	0.21
Intravenous nitroglycerin	152 (61%)	109 (62%)	43 (59%)	0.78
Inotropic therapy	39 (16%)	23 (13%)	16 (22%)	0.09
IABP	20 (8%)	13 (7%)	7 (10%)	0.61
Killip class ≥2 on admission	103 (41%)	54 (31%)	49 (67%)	<0.001

*Angiography was performed in 216 patients (86%): 157 patients with E/e′ ≤15 and 59 patients with E/e′ >15. Data are expressed as the mean value ± SD or number (percentage) of patients.

CABG = coronary artery bypass grafting; IABP = intra-aortic balloon pump; MI = myocardial infarction; PCI = percutaneous coronary intervention.

Statistical analyses. Continuous data are expressed as the mean value ± SD and compared using the Mann-Whitney *U* test, unless otherwise specified. Categorical data are presented as absolute values and percentages and compared using the Fisher exact test. Correlations were calculated using the Spearman rho test, and independent predictors of an elevated E/e′ ratio were identified by regression analyses. Survival was plotted according to the Kaplan-Meier method, and mortality rates were compared using the log-rank test. Further estimations of risk were performed using Cox proportional hazard models. Potential independent predictors of outcome were identified by univariate analyses. All univariate predictors were then entered in a stepwise manner into a multivariable model of survival, with entry and retention set at a significance level of <0.05. Where appropriate, probability values take into account multiple comparisons. SPSS version 9.0 (SPSS Inc., Chicago, Illinois) was used for all analyses.

Table 2. Echocardiographic Characteristics

Characteristic	All Patients (n = 250)	E/e′ ≤15 (n = 177)	E/e′ >15 (n = 73)	p Value
Timing of echocardiogram*	1.6 ± 1.6	1.7 ± 1.5	1.5 ± 1.6	0.23
LVEF (%)	48 ± 13	50 ± 12	42 ± 14	<0.001
>40%	178 (71%)	141 (80%)	37 (51%)	
≤40%	72 (29%)	36 (20%)	36 (49%)	<0.001
LV end-diastolic dimension (mm)	50 ± 5	50 ± 5	51 ± 7	0.42
LV end-systolic dimension (mm)	37 ± 9	35 ± 8	41 ± 9	0.01
Wall motion score index	1.6 ± 0.4	1.5 ± 0.4	1.8 ± 0.4	<0.001
Peak E-wave velocity (m/s)	0.8 ± 0.2	0.7 ± 0.2	1.0 ± 0.2	<0.001
Peak A-wave velocity (m/s)	0.8 ± 0.2	0.7 ± 0.2	0.8 ± 0.3	0.06
Pulmonary vein systolic/diastolic ratio	1.3 ± 1.1	1.4 ± 1.2	1.0 ± 0.5	<0.001
E/A ratio	1.2 ± 0.6	1.1 ± 0.4	1.5 ± 0.9	0.001
Deceleration time† (ms)	201 ± 52	206 ± 45	188 ± 65	<0.001
≤140	21 (9%)	6 (4%)	15 (22%)	
>140	219 (91%)	165 (96%)	54 (79%)	<0.001
Mitral annulus e′ (cm/s)	6.51 ± 2.21	7.27 ± 2.09	4.67 ± 1.13	<0.001
E/e′	13.8 ± 6.8	10.5 ± 2.8	22.0 ± 6.8	<0.001
Moderate or severe MR‡	34 (14%)	15 (9%)	19 (26%)	0.001

*Days after admission. †Deceleration time could be assessed in 240 patients (96%). ‡Mitral regurgitation could be graded in 242 patients (97%). Data are expressed as the mean value ± SD or number (percentage) of patients.

DT = deceleration time; LV = left ventricular; LVEF = left ventricular ejection fraction; MR = mitral regurgitation.
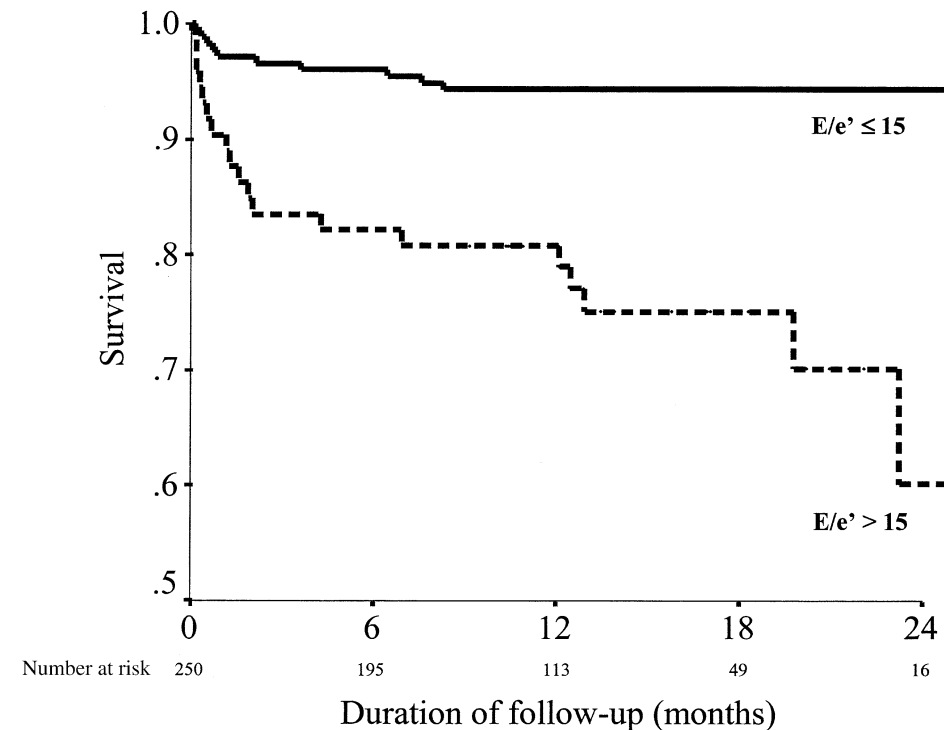
Figure 2. Kaplan-Meier plot demonstrating survival in patients classified according to an E/e′ ratio of ≤15 or >15.

RESULTS

The study cohort consisted of 155 male (68%) and 95 female (32%) patients with a mean age of 68 ± 13 years (Table 1). Baseline echocardiograms were obtained 1.6 ± 1.6 days after admission. The echocardiographic characteristics of the entire study cohort and of those patients with E/e′ ≤15 and >15 are shown in Table 2.

Seventy-three patients (29%) had an E/e′ >15. These patients were older and more commonly female (Table 1). In addition, they more often had diabetes mellitus and a history of MI and were more likely to present with clinical evidence of LV failure, as determined by the Killip class on admission. There were moderate correlations between the E/e′ ratio and Killip class (r = 0.43, p < 0.001) and mitral DT (r = −0.3, p < 0.001). The only independent clinical predictor of an E/e′ ratio >15 was Killip class ≥2 on admission (odds ratio 4.65, 95% confidence interval [CI] 2.59 to 8.34, p < 0.001).

On echocardiography, patients with an E/e′ ratio >15 had worse systolic function (as determined by the EF and WMSI) and a shorter DT (Table 2). They were also more likely to have moderate or severe mitral regurgitation.
Predictors of outcome. Vital status data were obtained for all subjects. During a median follow-up period of 13 months (interquartile range 11 to 19), 29 patients (12%) died. The majority of deaths (19/29 [66%]) occurred in patients with an E/e′ ratio in the upper three deciles (corresponding to those individuals with a ratio >15). Figure 2 displays

survival in patients dichotomized according to E/e′ >15, confirming the excess mortality associated with an elevated ratio (log-rank statistic 21.3, p < 0.0001). Similar analyses were also performed after stratifying patients on the basis of their LVEF. An E/e′ ratio >15 predicted decreased survival both in patients with an EF >40% (n = 178, log-rank statistic 12.3, p = 0.0005) and ≤40% (n = 72, log-rank statistic 3.93, p = 0.048) (Fig. 3). When patients were stratified on the basis of their DT, an E/e′ ratio >15 was a powerful predictor of mortality in patients with a DT >140 ms (n = 219, log-rank statistic 10.7, p = 0.001). In patients with a DT ≤140 ms (n = 21), the prognostic utility of an E/e′ ratio >15 did not attain statistical significance (log-rank statistic 3.3, p = 0.07). However, none of the six patients with a DT ≤140 ms, but E/e′ ≤15, died during follow-up, compared with 6 of the 15 with a DT ≤140 ms, but E/e′ >15. An E/e′ ratio >15 also predicted survival, regardless of whether the patient had ST-segment elevation (n = 120, log-rank statistic 18.46, p < 0.0001) or non–ST-segment elevation MI (n = 130, log-rank statistic 5.51, p = 0.02).

Univariate predictors of outcome are shown in Table 3. In a stepwise multivariable model in which all univariate (p < 0.05) predictors of outcome were considered, the most powerful independent prognostic indicator was an E/e′ ratio >15 (risk ratio [RR] 4.8, 95% CI 2.1 to 10.8, p = 0.0002). The other independent predictors were myocardial revascularization during the index admission (RR 0.31, 95% CI
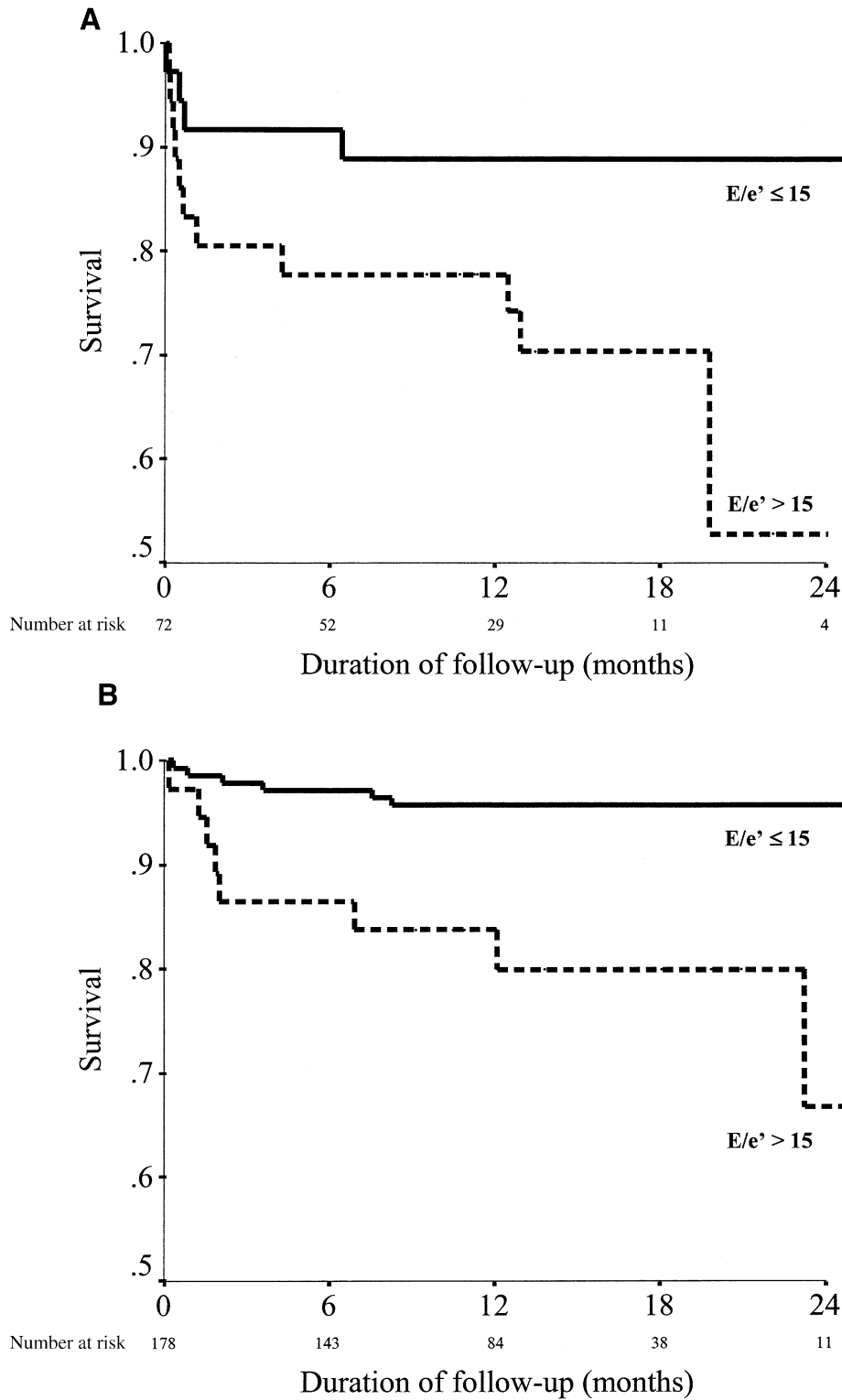
Figure 3. Survival of patients with an E/e′ ratio of ≤15 or >15, stratified according to left ventricular ejection fraction: **(A)** patients with ejection fraction ≤40%; **(B)** patients with ejection fraction >40%.

0.14 to 0.69, p = 0.004) and the index MI being anterior (RR 2.24, 95% CI 1.01 to 4.98, p = 0.047).

The incremental value of an E/e′ ratio >15 is shown in

Figure 4. The addition of E/e′ >15 significantly improved the prognostic utility of a model containing clinical variables (age, Killip class ≥2 on admission, anterior MI, myocardial

Table 3. Univariate Predictors of All-Cause Mortality

Characteristic	Survivors (n = 221)	Deceased (n = 29)	Risk Ratio (95% CI)	p Value
Age (yrs)	67.7 ± 13.4	72.4 ± 11.5	1.02 (0.99–1.05)†	0.16
Male gender	136 (62%)	19 (66%)	1.17 (0.54–2.52)	0.69
Anterior MI	89 (40%)	17 (59%)	1.93 (0.92–4.05)	0.08
ST-segment elevation MI	108 (49%)	12 (41%)	0.77 (0.37–1.60)	0.48
Multivessel disease*	104 (54%)	13 (57%)	1.13 (0.50–2.59)	0.76
PCI or CABG during index admission	160 (72%)	15 (52%)	0.45 (0.22–0.93)	0.03
Killip class ≥2 on admission	84 (38%)	19 (66%)	3.09 (1.43–6.65)	0.004
Diabetes mellitus	50 (23%)	6 (21%)	0.88 (0.36–2.15)	0.77
Hypertension	128 (58%)	14 (48%)	0.70 (0.34–1.45)	0.34
Previous MI	42 (19%)	8 (28%)	1.55 (0.68–3.49)	0.30
Current smoker	51 (23%)	6 (21%)	0.91 (0.37–2.23)	0.84
LVEF (%)	49.1 ± 12.6	40.1 ± 15.2	0.95 (0.93–0.98)‡	0.0004
Wall motion score index	1.57 ± 0.42	1.79 ± 0.49	2.82 (1.28–6.22)	0.01
Deceleration time				
≤140 ms	15 (7%)	6 (23%)	3.53 (1.41–8.81)	0.007
>140 ms	199 (93%)	20 (76%)	0.28 (0.11–0.71)	0.007
Moderate or severe MR	26 (12%)	8 (29%)	2.7 (1.2–6.2)	0.02
Peak E-wave velocity (m/s)	0.80 ± 0.23	0.89 ± 0.30	3.59 (0.85–15.1)§	0.08
Mitral annulus e′ (cm/s)	6.65 ± 2.18	5.45 ± 2.18	0.74 (0.61–0.91)‖	0.005
E/e′	13.3 ± 6.4	17.8 ± 8.0	1.07 (1.03–1.10)	0.0005
E/e′ >15	54 (24%)	19 (66%)	5.06 (2.35–10.9)	<0.0001

*Angiography was performed in 216 (86%) patients, of whom 193 survived and 23 died during follow-up. †Risk ratio per year. ‡Risk ratio per 1% increase in ejection fraction. §Risk ratio per 1 m/s increase in mitral peak E-wave velocity. ‖Risk ratio per 1 cm/s increase in mitral annulus e′ velocity. Data are expressed as the mean value ± SD or number (percentage) patients.
Abbreviations as in Tables 1 and 2.

revascularization during admission) and conventional echocardiographic indexes of LV systolic and diastolic function (EF and DT).

Discharge medication and outcome. At hospital discharge, there was no difference in the use of aspirin (65/68 vs. 169/174, p = 0.69) and angiotensin-converting enzyme inhibitors/angiotensin II antagonists (47/68 vs. 105/174, p = 0.24) in patients with E/e′ >15 and ≤15, respectively. In contrast, patients with an E/e′ >15 were more likely to be discharged on diuretic therapy (31/68 vs. 22/174, p < 0.001) and less likely to be prescribed beta-blockers (51/68

Figure 4. Incremental value of an E/e′ ratio >15 in predicting all-cause mortality. The addition of left ventricular ejection fraction (LVEF), deceleration time (DT), and E/e′ resulted in significant incremental improvements in the predictive value of a model including clinical variables (age, Killip class ≥2 on admission, anterior myocardial infarction, and myocardial revascularization during the index admission): chi-square = 20.8 with 4 degrees of freedom for clinical factors; chi-sqaure = 28.8 with 5 degrees of freedom for clinical factors plus LVEF; chi-square = 33.2 with 6 degrees of freedom for clinical factors plus LVEF plus DT ≤140 ms; and chi-square = 43.0 with 7 degrees of freedom for clinical factors plus LVEF plus DT ≤140 ms plus an E/e′ ratio <15.

vs. 153/174, p = 0.02) and lipid-lowering drugs (44/68 vs. 142/174, p = 0.007).

Discharge on diuretics was associated with an increased risk of death (RR 3.8, 95% CI 1.6 to 8.9, p = 0.003). Conversely, patients discharged on lipid-lowering drugs and/or beta-blockers were less likely to die during follow-up (RR 0.3, 95% CI 0.1 to 0.6, p = 0.002 and RR 0.4, 95% CI 0.1 to 0.9, p = 0.03, respectively). Being discharged on aspirin and/or angiotensin-converting enzyme inhibitors/angiotensin II antagonists had no significant effect on survival in this cohort.

In the 242 patients (97%) who survived to hospital discharge, an E/e′ ratio >15 remained a powerful predictor of death (RR 5.5, 95% CI 2.2 to 13.6, p < 0.001). It remained an independent predictor when entered into a stepwise multivariate model that also included discharge on diuretics, discharge on lipid-lowering drugs, and discharge on beta-blockers (RR 3.7, 95% CI 1.4 to 9.3, p = 0.006). The other independent predictors in this model were discharge on diuretics (RR 2.8, 95% CI 1.2 to 6.9, p = 0.02) or lipid-lowering drugs (RR 0.3, 95% CI 0.1 to 0.7, p = 0.006).

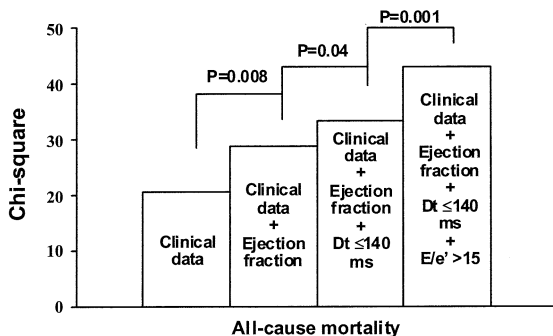## DISCUSSION

The principal finding of the current study was that the E/e′ ratio was a powerful predictor of survival after acute MI. An E/e′ ratio of >15 proved to be superior, in this respect, to other clinical or echocardiographic features measured in this study. Furthermore, it provided prognostic information incremental to these parameters.

Left ventricular filling pressure and survival after acute MI. Elevated PCWP is associated with a higher mortality rate after acute MI (20–22). There are several potential explanations for this. Higher LV filling pressures are usually indicative of larger infarcts with more severe systolic dysfunction (1,23–26). In addition, LV pressure overload predisposes to ventricular remodeling, neurohormonal activation, and increased excitability (4,27,28), all of which would be expected to adversely affect the outcome.

Despite its prognostic value, the measurement of PCWP has obvious drawbacks (29–33). In contrast, Doppler echocardiographic assessment of transmitral flow provides a noninvasive means of identifying patients with elevated left atrial pressures (34,35). Mild diastolic dysfunction is characterized by impaired relaxation of the LV (without elevation of LV filling pressures). This means that the ventricle takes longer to fill (lengthening the DT), with an increased reliance on the atrial component of diastolic filling (reducing the E/A ratio). Worsening diastolic function is associated with rising left atrial pressures. This results in a higher early diastolic gradient across the mitral valve, with rapid equalization of the pressures in the left atrium and ventricle. Initially, this normalizes the DT and mitral E/A ratio (pseudonormalization), but ultimately the mitral E wave becomes markedly predominant and the DT becomes very abbreviated (35).

Advanced diastolic dysfunction is associated with an adverse outcome after acute MI, with an abbreviated DT being particularly predictive (2–6). The current data confirm the prognostic value of a short DT. In addition, they corroborate the well-documented prognostic value of clinical indicators of LV filling pressures, such as Killip class (21).

Added value of E/e′. The current study demonstrates that, in the acute setting, elevated E/e′ is moderately correlated with traditional transmitral Doppler evidence of elevated LV filling pressures, but is a more powerful prognostic indicator. This is in keeping with previous data demonstrating that E/e′ is better correlated with invasive measurement of mLVEDP (7). E/e′ was also moderately correlated with Killip class on admission but, once more, proved to be a superior predictor of survival. Again, this might be expected given the limited correlation between clinical features of elevated LV filling pressures and invasive hemodynamics (36,37), with the latter having prognostic superiority (37).

Transmitral diastolic flow velocities and DT correlate well with LV filling pressure in patients with impaired LV systolic function but are of limited value in patients with preserved LV systolic function (7,38). In contrast, the E/e′ ratio correlates well with filling pressure, even in patients with a normal LVEF (7). In the current cohort, an E/e′ ratio >15 was a significant predictor of an adverse outcome, regardless of LVEF (Fig. 3), the presence or absence of ST-segment elevation, or drug therapy on hospital discharge.

The E/e′ ratio was superior to conventional parameters of

LV systolic function, such as LVEF and WMSI, for prediction of prognosis. However, it is important to recognize that measurement of E/e′ provides complementary prognostic data, with the maximum information obtained by combining this with clinical, systolic, and conventional diastolic parameters.

Study limitations. Not all patients presenting with acute MI during the study period underwent echocardiography. In addition, measurement of E/e′ was performed at the discretion of the sonographer and physician performing echocardiography. Nevertheless, the current cohort represents a consecutive series of patients in whom this measurement was available. Thus, it seems unlikely that a selection bias could have influenced the observed results, which may affect the findings' applicability to other populations. Conversely, the measurements were obtained by multiple observers working in a clinical environment, suggesting that the findings may be widely applicable.

Like all currently utilized Doppler, clinical, and hemodynamic indexes, the E/e′ ratio reflects an instantaneous measure of LV filling pressures. These may change over the course of the peri-infarct period, and a single measurement may not convey maximal prognostic information.

Conclusions. The current data demonstrate that an E/e′ ratio of >15 is a powerful predictor of decreased survival after acute MI. The prognostic value of E/e′ was incremental to clinical factors and conventional echocardiographic parameters of LV systolic and diastolic function. Measurement of E/e′ may therefore assist in the risk stratification of patients in this setting.

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Noninvasive estimation of left ventricular filling pressure by e/e' is a powerful predictor of survival after acute myocardial infarction

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