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Evaluation and prognostic significance of left ventricular diastolic function assessed by Doppler echocardiography in the early phase of a first acute myocardial infarction

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Aim To study the prognostic significance of left ventricular diastolic function evaluated by transmitral and pulmonary venous flow velocities obtained in the early phase of a first acute myocardial infarction in relation to later development of congestive heart failure.

Methods Pulsed Doppler echocardiography of transmitral and pulmonary venous flow was assessed in 65 consecutive patients with a first myocardial infarction within 1 h of arrival in the coronary care unit.

Results A univariate regression analysis identified age, left ventricular ejection fraction ≤45%, mitral E deceleration time ≤130 ms, E/A ratio >1·5, peak pulmonary venous atrial flow velocity ≥30 cm . s^{-1} and a difference between mitral and pulmonary venous atrial flow duration <0 ms as variables significantly related to the development of congestive heart failure. However, in a multivariate analysis only mitral E deceleration time ≤130 ms and age were significant independent variables related to the development of congestive heart failure during the first week following a first acute myocardial infarction.

Conclusion Assessment of left ventricular diastolic function complements measurements of systolic function in the evaluation of cardiac function, and mitral deceleration ≤130 ms best identifies patients at risk of development of congestive heart failure following acute myocardial infarction.

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Key Words: Prognosis, diastolic function, Doppler echocardiography, MI

Introduction

Clinical evidence of heart failure in acute myocardial infarction is associated with increased mortality even if the manifestations of failure resolve within the first 24 h[1–3]. Left ventricular systolic function after acute myocardial infarction has been extensively studied in relation to the development of heart failure and mortality[4–6]. However, it has become increasingly apparent that left ventricular diastolic function contributes to signs and symptoms of clinical heart failure[7–12]. In post-infarction patients, left ventricular systolic and diastolic dysfunction frequently coexist even though both isolated systolic and diastolic dysfunction can be identified[13–15]. Treatment with angiotensin enzyme converting inhibitors (ACE inhibitors) is associated with improved survival in patients with left ventricular systolic dysfunction and congestive heart failure after acute myocardial infarction[16,17]. However, the impact of early assessment of diastolic function in the very early phases of acute myocardial infarction is poorly investigated and left ventricular diastolic function has not been taken into consideration in these trials[15,20–22]. Moreover, the optimal time to start ACE inhibitors and the optimal selection of patients with acute myocardial infarction remains to be established[16–20].

Based on echocardiographic Doppler variables obtained from mitral inflow, three main left ventricular diastolic filling patterns are identified: normal, impaired relaxation and restrictive filling patterns[23–25]. Impaired left ventricular relaxation, characterized by a prolonged isovolumetric relaxation time and a deceleration time characterized by early diastole with dominant atrial and reduced filling velocities, is usually associated with normal or near normal diastolic filling pressures[26]. The

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restrictive filling pattern is characterized by a short isovolumetric relaxation time, a reduced deceleration time, enhanced early filling and decreased filling during atrial systole. The restrictive filling pattern, which is associated with decreased left ventricular compliance in the presence of elevated filling pressure, is often seen in patients with advanced cardiac disease[25]. If both impaired relaxation and decreased compliance of the ventricle is present at the same time, the abnormalities will have opposing effects on the left ventricular filling pattern. The balance between early and late diastole will appear normal or more accurately 'pseudonormal'[25]. Assessment of mitral deceleration time in early filling has provided important information about filling pressures and is useful, particularly in patients with a pseudonormal mitral flow velocity pattern[13]. However, mitral flow velocity is dependent on a complex sequence of interrelated events and influenced by many factors, including heart rate, age, loading conditions and valvular regurgitation. Therefore, caution is needed when assessing mitral flow alone to evaluate left ventricular diastolic function[25,33–37]. Recently, assessment of pulmonary venous flow velocities, obtained by transthoracic recording, has been useful in complementing the information obtained from mitral flow velocities and improving the assessment of left ventricular diastolic pressures[33–41].

The aims of this study were, firstly to characterize left ventricular diastolic function evaluated by transmitral and pulmonary venous flow velocities, and secondly to study the prognostic significance of Doppler measurements in relation to the development of heart failure in the early phase of a first acute myocardial infarction.

Methods

Study population

This prospective study consisted of 65 consecutive patients admitted to the coronary care unit with a first acute myocardial infarction defined according to the WHO criteria with two out of the following three characteristics (1) typical chest pain, (2) electrocardiographic evidence of myocardial infarction (ST elevation >1 mm in contiguous leads or subendocardial injury pattern) and (3) transient elevation of creatinine kinase MB $\geq 20\ U \cdot l^{-1}$ (normal $\leq 6\ U \cdot l^{-1}$). Two patients were excluded for reasons mentioned below. All patients were between 40 to 75 years of age. No patient had significant valvular heart disease and all were in sinus rhythm. Patient demographics are shown in Table 1.

An echocardiographic examination was performed within 1 h of arrival in the coronary care unit by the same examiner (S.H.P.) except for two investigations (S.E.J.). On the basis of the presence of clinical heart failure during the first week of hospitalization, the patients were divided into two groups. Group A patients had no sign of heart failure (Killip class I) and group B patients had heart failure (Killip class II–IV). All patients' clinical status were evaluated daily during hospitalization including heart failure status.

The study was approved by the local scientific ethical committee and written informed consent was given by each participant.

Table 1 Patient characteristics (n=63)

Age (years)	61 ± 10
Men	56 (89)
Diabetes mellitus	5 (8)
Systemic hypertension	10 (16)
Total se-cholesterol (mmol . l^{-1})	6·6 ± 1·1
Smoker, incl. previous (%)	81
Anterior MI	25 (40)
Inferior MI	30 (47)
Indeterminate MI	8 (13)
Q wave MI	44 (70)
Peak CK-MB	128 ± 114
Thrombolytic therapy	47 (75)
Time (h) from chest pain onset to arrival to CCU	3·7 ± 3·8

Values expressed as number (%) or mean ± SD. MI=myocardial infarction.

Echocardiography

Two-dimensional and Doppler echocardiographic examinations were performed with an ATL Ultramark 7 cardiac ultrasound unit using a 2·5 MHz transducer. Blood pressure and heart rate were obtained simultaneously. All two-dimensional data were stored digitally whereas pulsed Doppler measurements were stored on high quality video tape for later analysis using a Panasonic AG-7350 video cassette recorder. Left ventricular volumes and ejection fraction were evaluated from standard apical two and four chamber views using Simpson's biplane disc method[42]. Left ventricular volumes were corrected for body surface area and the mean of three measurements were used. Left ventricular diastolic function was evaluated by the pulsed Doppler technique, measuring mitral and pulmonary venous flow. Mitral flow velocities were obtained from the apical four-chamber view by placing the sample volume between the tips of the mitral leaflets, and pulmonary venous flow velocities were obtained by placing the sample volume at least 1 cm into the right superior pulmonary vein. Each Doppler profile was analysed by digital tracing and Doppler measurements were calculated from an average of five consecutive cardiac cycles. Mitral inflow was applicable in 63 patients and two patients were excluded as the mitral flow was not measurable due to fusion of the Doppler waves. Pulmonary venous flow was obtained in 80% of all (50/63 patients) cases. The following transmitral and pulmonary venous Doppler parameters were analysed: (1) mitral flow parameters — peak early (E) and late (A) transmitral filling velocities, their ratio (E/A ratio) and

Table 2 Clinical and two-dimensional echocardiographic data in patients with MI according to their left ventricular filling pattern

	Group I (n=24)	Group II (n=23)	Group III (n=16)
Age (years)	59 ± 11	65 ± 6*	60 ± 10†
Heart rate (beats . min^{-1})	74 ± 9	74 ± 9	77 ± 10
SBP (mmHg)	130 ± 17	146 ± 37	130 ± 18
DBP (mmHg)	77 ± 15	87 ± 20	84 ± 14
Peak CKB (U . l^{-1})	115 ± 18	107 ± 114	172 ± 112
Anterior MI	8	6	11‡,†
Inferior/indeterminate MI	13/3	11/6	5/0
Q/non-Q wave MI	15/9	17/6	12/4*
LA (mm)	37 ± 5	37 ± 5	37 ± 5
MSS (mm)	8 ± 3	10 ± 4*	14 ± 5‡‡‡,†
EDVI (ml . m^{-2})	67 ± 19	73 ± 23	74 ± 22
ESVI (ml . m^{-2})	33 ± 10	39 ± 18	46 ± 7‡‡,†
EF (%)	50 ± 10	48 ± 9	38 ± 11‡‡‡,††

Values are expressed as mean ± SD unless otherwise noted.
SBP=systolic blood pressure; DBP=diastolic blood pressure; MI=myocardial infarct; LA=left atrium; MSS=mitral septal separation; EDVI=end-diastolic volume index; ESVI=end systolic volume index; EF=ejection fraction.
*P<0·05, **P<0·01, ***P<0·001 group I vs II, ‡P<0 05, ‡‡P<0·01, ‡‡‡P<0·001 group I vs III, †P<0·05, ††P<0·01, †††P<0·001 group II vs III.

the deceleration time of the E velocity. The isovolumetric relaxation time, defined as the time from aortic valve closure to mitral valve opening, was assessed by simultaneously measuring the flow into the left ventricular outflow tract and mitral inflow by Doppler echocardiography[23,24]. (2) pulmonary venous flow–peak pulmonary venous flow velocity during ventricular systole (S), peak pulmonary venous flow velocity during ventricular diastole (D), peak reverse pulmonary venous flow velocity associated with atrial contraction (PVA), duration of pulmonary reversal flow during atrial contraction (PVAD), velocity time integral of systolic and diastolic wave (S-VTI, D-VTI), velocity time integral of systolic flow divided by total velocity integral of systolic and diastolic flow (PVSF), difference between the duration of the mitral A-wave and the pulmonary reversal wave (AD-PVAD)[25,38]. All echocardiographic measurements were analysed without any knowledge of the patients' clinical data. Inter- and intra-observer variability analysis was performed in 15 randomly chosen patients and was found to be less than 5% for all echocardiographic variables. The time interval for the inter-observer variability analysis was less than 30 min.

According to the Doppler transmitral flow velocity profile, as expressed by the mitral E deceleration time and the measurement of the isovolumetric relaxation time, the study patients were assigned to the following three groups: Group I, with a mitral E deceleration time >140 ms and an isovolumetric relaxation time <100 ms representing a normal filling pattern (24 patients, 38%), group II, with a mitral E deceleration time >140 ms and an isovolumetric relaxation time ≥100 ms considered to be an expression of impaired relaxation (23 patients, 37%), group III, with a deceleration time ≤140 ms, which may signify a pseudonormal or restrictive filling pattern (16 patients, 25%)[24,43].

Statistical analysis

All results are expressed as mean ± one standard deviation. Comparisons between groups were made by a two-sample t-test or the Mann–Whitney rank test for unpaired data when tests for normal distribution failed. The chi-square test was used for dichotomous data. Uni- and multivariate logistic regression analysis was performed to identify independent correlates for the presence of heart failure (Killip >1) using the SAS (Statistics Analysis System) statistical program, P values <0·05 were considered to be significant.

Results

Clinical characteristics and two-dimensional echocardiographic data

The clinical and echocardiographic data of the groups are presented in Table 2. Patients in group III had a significantly higher end systolic volume index, increased mitral septal separation and lower ejection fraction than patients in groups I and II. Patients in group III more commonly had anterior Q-wave infarcts than patients in groups I and II. Patients in group II were significantly older than patients in groups I and II. No difference between groups was found in relation to heart rate, systolic and diastolic blood pressure, creatine kinase fraction B and size of left atrium.

Diastolic Doppler variables

The transmitral and pulmonary venous Doppler measurements of the groups are presented in Table 3. As expected, significant differences were found in the mitral

Table 3 Transmitral and pulmonary Doppler variables in patients grouped according to their left ventricular filling pattern

	Group I (n=24)	Group II (n=23)	Group III (n=16)
E (cm . s^{-1})	62 ± 14	46 ± 14	66 ± 11†††
A (cm . s^{-1})	60 ± 17	69 ± 17	47 ± 17‡‡‡,†††
E/A ratio	1·1 ± 0·3	0·7 ± 0·3***	1·67 ± 0·8‡‡,†††
DT (ms)	185 ± 35	204 ± 38*	111 ± 16‡‡‡,†††
IRT (ms)	74 ± 11	117 ± 11***	56 ± 17‡‡‡,†††
S (cm . s^{-1})	44 ± 9	41 ± 9	34 ± 9‡,†
D (cm . s^{-1})	36 ± 11	34 ± 11	43 ± 11†
S-VTI	11·7 ± 2·1	9·8 ± 3·2*	− 6·1 ± 4·1‡‡,†
D-VTI	8·8 ± 3·1	7·5 ± 2·7	8·6 ± 2·5
S/D ratio	1·27 ± 0·25	1·29 ± 0·31	0·84 ± 0·44‡‡,††
PVSF (%)	0·58 ± 0·08	0·57 ± 0·12	0·40 ± 0·14‡‡‡,††
PVA (cm . s^{-1})	26 ± 3	27 ± 4	28 ± 3
AD-PVAD (ms)	21 ± 28	10 ± 24	− 20 ± 19‡‡‡,†††

Values expressed as mean ± SD.
E=peak flow velocity during early diastole; A=peak flow velocity at atrial contraction; DT=deceleration time of early mitral flow; IRT=isovolumetric relaxation time; S=peak systolic pulmonary venous flow velocity; D=peak diastolic pulmonary venous flow velocity; PVSF=systolic fraction of pulmonary venous flow calculated from the velocity time integrals of S and D; PVA=peak pulmonary venous A wave velocity; AD-PVAD=duration of mitral A wave minus the duration of pulmonary venous A wave. For *P* value symbols, see footnote to Table 2.

E deceleration time and isovolumetric relaxation time between the groups due to the group criteria. However, patients in group II had significantly lower E, higher A, and a lower E/A ratio compared to patients in group I. Their pulmonary venous flow velocities and durations were similar except from the systolic velocity time integral. Both the transmitral and pulmonary venous flow velocities and durations were significantly different in group III compared to groups I and II. The peak flow velocity at atrial contraction was decreased and the E/A ratio increased in group III compared to groups I and II. The peak systolic pulmonary flow velocity, the systolic pulmonary venous time integral and the pulmonary systolic flow velocity time integral fraction were significantly decreased in group III. In addition, pulmonary venous flow reversal duration exceeded the duration of the mitral A wave in group III, which was significantly different from groups I and II.

Isolated left ventricular systolic dysfunction (defined as an ejection fraction <50% and a normal filling pattern) was found in 13 patients (21%). Left ventricular diastolic dysfunction (defined as impaired relaxation or a pseudonormalized or restrictive filling pattern, see our criteria) and a normal systolic function were found in 15 patients (24%). Combined systolic and diastolic dysfunction was found in 24 patients (38%) and combined normal systolic and diastolic function in 11 patients (17%).

Congestive heart failure following acute myocardial infarction

Clinical characteristics

Of the 63 patients studied, 41 had no sign of heart failure (group A) and 22 patients were in Killip class II to IV (group B) on admission or during the first week of hospitalization. Eight of these patients had signs of heart failure within the first 24 h of arrival and 14 patients developed heart failure on days 2–7 after arrival in the CCU. There was no statistically significant difference between groups A and B regarding diabetes mellitus, history of hypertension, infarct location, Q wave infarct, peak creatine kinase B, given thrombolytic therapy and systolic blood pressure. Heart rate was higher in group B but did not reach a significant level ($P<0·07$). However, group B was significantly older than group A (Table 4).

Left ventricular systolic and diastolic echocardiographic data

The left ventricular end-diastolic and systolic volume index was significantly larger in patients with congestive heart failure compared to patients free of heart failure. The left ventricular ejection fraction was significantly lower in group B than in group A (Table 4). No significant difference was found in E/A ratio between the groups. The deceleration time of the mitral E wave was significantly shorter in group B compared to group A. The isovolumetric relaxation time was not significantly different between the groups.

No significant difference was found concerning the pulmonary venous systolic and diastolic peak velocities between patients in groups A and B. However, pulmonary venous reversal peak flow velocity was significantly greater in group B than group A. Furthermore the difference between mitral A wave duration and pulmonary venous reversal wave duration was significantly lower (negative) in group B compared to group A. There was no significant difference between groups concerning pulmonary systolic venous flow velocity time integral fraction.

Table 4 Relation of echocardiographic Doppler variables to development of congestive heart failure following MI

	Group A (n=41)	Group B (n=22)	*P* value
Age (years)	59 ± 10	65 ± 9	0·01
Heart rate (beats . min^{-1})	71 ± 11	77 ± 13	ns
EDVI (ml . m^{-2})	67 ± 19	79 ± 24	0·05
ESVI (ml . m^{-2})	34 ± 18	47 ± 19	0·01
EF (%)	50 ± 10	41 ± 10	0·001
E/A ratio	1·0 ± 0·4	1·2 ± 0·8	ns
DT (ms)	188 ± 43	150 ± 49	0·003
IRT (ms)	91 ± 28	78 ± 28	ns
S (cm . s^{-1})	41 ± 10	39 ± 10	ns
D (cm . s^{-1})	36 ± 11	40 ± 11	ns
PVSF (%)	0·55 ± 0·12	0·49 ± 0·17	ns
PVA (cm . s^{-1})	26 ± 4	29 ± 3	0·003
AD-PVAD (ms)	14 ± 28	−6 ± 28	0·02

Values expressed as mean ± SD. For abbreviations see previous tables. Statistics between groups. ns=not significant.

Congestive heart failure and left ventricular filling patterns

No patients in group I with normal diastolic Doppler variables developed clinical heart failure. Eleven had a preserved left ventricular ejection fraction (≥50%) whereas 13 patients had ejection fraction below 50%. In group II with impaired relaxation characteristics, 13 patients had a preserved ejection fraction whereas 10 patients had an ejection fraction below 50%. Fourteen patients developed clinical heart failure of whom 11 suffered from impaired relaxation of the left ventricle (42%); seven patients had a preserved ejection fraction. In group III with restrictive left ventricular filling, 11 patients (69%) developed clinical heart failure, of whom two had a preserved ejection fraction. Overall, five patients with clinical heart failure had preserved left ventricular ejection fraction.

Univariate and multivariate logistic regression analysis

A univariate analysis identified seven variables, which correlates significantly with the development of congestive heart failure: mitral E deceleration time ≤130 ms ($P<0·006$), pulmonary venous reversal peak velocity flow ≥30 cm . s^{-1} ($P<0·009$), difference between mitral and pulmonary venous atrial flow duration ≤0 ($P<0·05$), age ($P<0·01$), E/A ratio ≥1·5 ($P<0·05$), left ventricular ejection fraction ≤45% ($P<0·01$) and end-systolic volume index ($P<0·05$). The mitral E deceleration time of all the left ventricular systolic and diastolic variables best correlates with the presence or development of clinical heart failure. Multivariate analysis demonstrated that age and mitral E deceleration time ≤130 ms correlate significantly ($P<0·01$ and $P<0·006$, respectively) with the development of clinical heart failure within the first week of an acute myocardial infarction. These two variables were independent of all clinical as well as all left ventricular systolic and diastolic ehocardiographic variables.

In Table 5, patients with signs of congestive heart failure following myocardial infarction are subdivided according to their mitral E deceleration time (> or ≤130 ms). As expected, a significant difference was found between groups regarding the mitral E deceleration time. However, patients with a short deceleration time (n=11) had an increased E/A ratio, a decreased A (43 ± 15 vs 79 ± 18, $P<0·0001$) and a shorter isovolumetric relaxation time than the rest. In addition, pulmonary venous flow differed as the pulmonary velocity time integral systolic fraction, the difference between mitral and pulmonary venous flow durations during atrial contraction, was decreased and the pulmonary venous reversal peak velocity was increased in patients with a mitral deceleration time ≤130 ms compared to patients with a higher deceleration time. Patients with a mitral E deceleration time ≤130 ms significantly more often had an anterior infarction compared with a deceleration time ≥130 ms ($P<0·01$). All patients with congestive heart failure and an impaired relaxation filling pattern were in Killip class II, whereas patients with congestive heart failure and a restrictive or pseudonormal filling pattern were in Killip class III–IV ($P<0·05$).

Discussion

Previous combined haemodynamic Doppler flow studies have demonstrated the utility of mitral and pulmonary venous flow velocities in the assessment of left ventricular relaxation, compliance and filling pressures[22,24,26,44,45]. Among several mitral flow parameters, early diastole (E) and late atrial (A) peak velocity, their ratio (E/A ratio), the isovolumetric relaxation time and mitral early deceleration time are the most commonly used to evaluate left ventricular diastolic filling and are shown best to correlate to left ventricular pressure[13,24,29]. Assessment of pulmonary venous flow has recently been shown to be helpful in the interpretation of mitral flow patterns and estimation of filling pressures. A systolic fraction of total pulmonary venous flow less than 40% in patients with a pseudonormal mitral

Table 5 Characteristics of CHF patients grouped according to their mitral deceleration time

	DT ≤130 ms (n=11)	DT >130 ms (n=11)	*P* value
Age (years)	63 ± 10	67 ± 6	ns
Heart rate (beats . min^{-1})	77 ± 14	76 ± 12	ns
MI localisation (anterior/other)	8/3	4/7	0·01
ESVI (ml . min^{-1} (m^2)	51 ± 13	44 ± 18	ns
EF (%)	38 ± 12	41 ± 8	ns
E/A	1·8 ± 0·8	0·7 ± 0·3	0·001
DT (ms)	110 ± 17	187 ± 37	0·001
IRT (ms)	55 ± 17	103 ± 14	0·001
S (cm . s^{-1})	35 ± 15	43 ± 8	ns
D (cm . s^{-1})	50 ± 7	31 ± 5	0·001
PVSF (%)	0·37 ± 0·17	0·60 ± 0·05	0·01
PVA (cm . s^{-1})	30 ± 3	29 ± 3	ns
AD-PVAD (ms)	$-20 \pm 20_{10 \pm 27}$	0·05	

Mean expressed as mean ± SD unless otherwise noted. Statistics between groups; **P*<0·05, ***P*<0·01, ****P*<0·001.

filling pattern indicates elevated filling pressure[38,45]. Additionally, pulmonary venous flow reversal exceeding the duration of the mitral A wave is also associated with increased filling pressure[38].

In the present study, both transmitral and pulmonary flow velocities were used to assess left ventricular diastolic function early after onset of symptoms in patients with a first acute myocardial infarction. Both myocardial relaxation and compliance are affected by ischaemia, but the predominantly diastolic abnormality is an impairment in relaxation. Abnormal relaxation is also seen in aged and hypertrophied hearts, whereas decreased compliance is seen most often in patients with advanced dilated cardiomyopathy, and restrictive and cardiac amyloidosis[28,31]. Abnormal myocardial relaxation has been produced by balloon inflation during percutaneous transluminal coronary angiography and is also reported to be present in the subacute phase of a myocardial infarction[14,15,45]. Decreased left ventricular compliance (or its chamber stiffness) has recently been described in patients 1–2 days after an acute myocardial infarction[20,22].

Patients with impaired left ventricular relaxation are often asymptomatic or display mild functional impairment at exercise, and often have normal or near normal filling pressures. In contrast, patients with restrictive filling characteristics due to decreased compliance have moderate to severe functional impairment and are associated with elevated filling pressures[25].

In the present study, 31% of the patients developed clinical heart failure, which is consistent with previous studies[12,16]. As expected from previous studies, left ventricular systolic function was significantly decreased in patients with congestive heart failure compared to those without congestive heart failure. However, five patients with features of congestive heart failure had a normal ejection fraction but an abnormal diastolic filling pattern. A similar incidence of congestive heart failure and a normal ejection fraction have previously been reported among patients referred to hospital with clinical heart failure[7–9]. Moreover, only patients with impaired relaxation and a pseudonormal or restrictive filling pattern developed clinical heart failure. Patients with a normal left ventricular filling pattern showed no signs of clinical heart failure despite having systolic dysfunction comparable with patients with impaired left ventricular relaxation.

It is remarkable that among the patients who developed congestive heart failure, the left ventricular ejection fraction was similarly impaired in the subgroup with impaired relaxation and in patients with restrictive or pseudonormal filling. However, the latter group had a significantly higher Killip classification. This finding is in accordance with studies in chronic heart failure where the restrictive filling pattern, with a shortened mitral E deceleration and isovolumetric relaxation time, is associated with more impaired functional status[28–32].

Pseudonormal or restrictive filling may reflect primary changes in the infarcted and ischaemic myocardium or simply the ventricle filling on a steeper portion of the pressure–volume curve[47,48]. In this study, these patients demonstrated early dilation and significantly larger end-systolic volumes compared with patients with impaired ventricular relaxation. This finding might, in part, be compensatory to normalize the elevated filling pressure, as indicated by the shortened mitral deceleration time, the low systolic fraction of the total pulmonary venous flow and the pulmonary venous reversal flow exceeding the duration of the mitral A wave. In the present study, it is likely that decreased left ventricular compliance is primarily due to changes in the myocardium with a second rise in the pressure–volume curve, as the study was performed in the very early phase of the myocardial infarction with ongoing myocardial ischaemia.

In the present study, age and mitral E deceleration time ≤130 ms independently identified patients at risk of developing congestive heart failure during the

first week after first acute myocardial infarction. Mitral E deceleration time has previously been shown to be independent of heart rate[20]. This is useful in patients with a pseudonormalized transmitral flow and is shown to possess prognostic information[22,28–32]. In patients with a mitral deceleration time below 140 ms, the systolic fraction of the total pulmonary venous flow was less than or equal to 40%, and the pulmonary venous reversal flow exceeded the duration of the mitral A wave, indicating an elevated filling pressure. Furthermore, as mitral E deceleration time and pulmonary venous flow velocities were significantly different in patients who developed congestive heart failure, compared with patients without congestive heart failure following acute myocardial infarction, these measures seem useful, complementing left ventricular systolic measures in patients with acute myocardial infarction. Left ventricular diastolic dysfunction is an important feature in patients developing congestive heart failure. Thus, early assessment of diastolic function may be helpful in selecting patients who might benefit from early intervention with ACE inhibition following acute myocardial infarction. Further studies are needed to demonstrate the importance of diastolic function and the effect of early intervention with ACE inhibitors on left ventricular diastolic function in patients with acute myocardial infarction.

Limitations

In this echocardiographic study, invasive haemodynamic measurements were not performed. However, systolic and diastolic variables have previously been correlated to simultaneous haemodynamic data. Pulmonary venous flow was only assessed transthoracically in about 80% of patients. This success rate is within the range of other Doppler studies and is considered acceptable, especially when the conditions under which these measurements are obtained are taken into consideration. Finally, this study was relatively small in the numbers of patients and our results should be confirmed by larger studies.

Conclusions

Left ventricular diastolic dysfunction is present early after onset of symptoms of a first acute myocardial infarction. Furthermore left ventricular diastolic function seems to play an important role in the development of clinical heart failure following acute myocardial infarction. Impaired relaxation of the left ventricle was the predominant diastolic filling abnormality (37%) but pseudonormal or restrictive filling patterns were also frequently present (25%) in patients with a first acute myocardial infarction. In addition, 24% of the patients had isolated diastolic dysfunction.

Development of congestive heart failure occurred in 31% of the patients during the first week after the acute event. In this group, early measurement of transmitral flow contained prognostic information that was superior to estimation of left ventricular ejection fraction. Patients with congestive heart failure had either impaired relaxation or pseudonormal/restrictive filling pattern in the left ventricle. However, patients with such patterns had features of significantly more severe congestive heart failure. Although most patients with heart failure had early systolic dysfunction with decreased ejection fraction, a subgroup (23%) had an abnormal filling pattern with preserved ejection fraction.

Patients at risk of developing congestive heart failure during the first week of a first acute myocardial infarction are best identified by a short mitral E deceleration time.

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