Journal of Cardiovascular Magnetic Resonance (2007) **9,** 877–882

ISSN: 1097-6647 print / 1532-429X online
DOI: 10.1080/10976640701693667

informa
healthcare

Tissue Cardiovascular Magnetic Resonance Demonstrates Regional Diastolic Dysfunction in Remote Tissue Early After Inferior Myocardial Infarction

Bernard P. Paelinck, MD, PhD,[1] Christiaan J. Vrints, MD, PhD,[1] Jeroen J. Bax, MD, PhD,[2] Johan M. Bosmans, MD, PhD,[1] Albert de Roos, MD, PhD,[3] and **Hildo J. Lamb, MD, PhD[3]**

Departments of Cardiology, University Hospital Antwerp, Edegem, Belgium,[1] Departments of Cardiology,[2] and Radiology,[3] Leiden University Medical Center, Leiden, The Netherlands

ABSTRACT

***Purpose*: To investigate regional diastolic and systolic function using tissue cardiovascular magnetic resonance (CMR), early after transmural myocardial infarction of the inferior wall due to single proximal right coronary artery disease. *Materials and Methods*: Velocity encoded CMR was used to measure early diastolic transmitral flow velocity (E), and regional, longitudinal, myocardial systolic (Sa) and early diastolic (Ea) velocities (tissue CMR) in 15 patients with a recent transmural inferior myocardial infarction and in 15 age and LV-mass index matched control subjects. An unpaired two-tailed *t* test was used to assess significance of continuous variables. *Results:* Global systolic (ejection fraction 46 $\pm$ 7% versus 57 $\pm$ 4%, p $=$ 0.000052) and global diastolic LV function (average Ea of infarcted or inferior, remote or anterior, adjacent or septal and lateral myocardium 6.8 $\pm$ 1.7 cm/s versus 10.4 $\pm$ 1.5 cm/s, p $=$ 0.0000012) were impaired in patients as compared to controls. Regional systolic and diastolic LV velocities were impaired in infarcted and adjacent tissue in patients. However, in remote or anterior tissue, systolic velocities were preserved (Sa 6.6 $\pm$ 2.0 cm/s versus 6.8 $\pm$ 1.4 cm/s, p $=$ 0.70), but diastolic velocities were impaired in patients as compared to controls (Ea 7.2 $\pm$ 2.3 cm/s versus 10.2 $\pm$ 2.5 cm/s, p $=$ 0.0026). *Conclusions*: Regional diastolic velocities early after inferior myocardial infarction are impaired in the infarcted, adjacent and remote tissue, but regional systolic velocities are preserved in remote tissue.**

INTRODUCTION

Impairment of global diastolic left ventricular (LV) function following myocardial infarction is related to LV remodeling (1) and is a predictor of cardiac death (2). Diastolic dysfunction is an early marker of disease. In myocardial infarction of the inferior wall, regional LV diastolic function, the relation between regional and global diastolic function, and the relation between regional diastolic and systolic function is largely unknown.

Myocardial infarction of the anterior wall causes impaired regional function in both infarcted, adjacent and remote myocardium (3, 4). Myocardial infarction of the inferior wall exhibits a smaller functional loss when compared to similar sized myocardial infarction of the anterior wall (5, 6). Therefore, we hypothesized that in myocardial infarction of the inferior wall, regional diastolic velocities of the remote tissue are impaired, while regional systolic velocities are preserved. Velocity encoded cardiovascular magnetic resonance (CMR) with low (30 cm/s) velocity encoding (tissue CMR) allows measurement of regional myocardial velocities (7–9).

Therefore, the purpose of the present study was to investigate regional diastolic and systolic function using tissue CMR, early after transmural myocardial infarction of the inferior wall due to single vessel proximal right coronary artery disease.

Received 10 May 2007; accepted 30 August 2007.
Keywords: Magnetic Resonance, Myocardial Infarction, Function, Remote.
Bernard P. Paelinck was supported by a grant of the Fund for Scientific Research Flanders, Brussels, Belgium.
Correspondence to:
Bernard P. Paelinck
Dept. of Cardiology
University Hospital Antwerp
Wilrijkstraat 10, 2650 Edegem
Belgium
tel: +32/3/8214182; fax: +32/3/8250848
email: Bernard.Paelinck@uza.be

METHODS

Subjects and study design

The study protocol was approved by the medical ethical committee, and all patients gave written informed consent before participation. Patients with a first acute q-wave myocardial infarction of the inferior wall and single proximal, dominant right coronary artery disease were included in the present study. All patients underwent percutaneous coronary intervention of the culprit lesion within 6 hours of the acute event and received treatment with beta-blockers and angiotensin-converting enzyme inhibitors. Patients with a contraindication for CMR, irregular heart rate, hypertension or diabetes mellitus were excluded. The data were compared with age and LV-mass index matched control subjects.

CMR measurements

CMR studies were performed using a Sonata MR scanner and a 12 channel Body Array surface coil (Siemens, Erlangen, Germany). The entire heart was imaged in the short-axis orientation with a breath-hold cine steady-state free precession technique (TrueFISP imaging) (9).

Late gadolinium enhancement (LGE) was studied 15 minutes after intravenous bolus injection of 0.2 mmol/kg gadolinium-DTPA (Magnevist, Schering, Berlin, Germany). The entire heart was imaged in short-axis orientation using a TrueFISP sequence (10).

Transmitral flow was acquired using a retrospective electrocardiographically triggered Flash phase-contrast CMR technique with a velocity sensitivity of 130 cm/s, as described before (9). In order to cover late diastolic filling, acquisition was performed throughout the cardiac cycle with a retrospective period of 1.2. Imaging parameters included the following: 30/3.2 (repetition time ms/echo-time ms), 5-mm section thickness, 240 × 256 matrix, 380 × 380 mm field of view, 1.6 × 1.5 mm in-plane spatial resolution and 30° flip angle. Two signals were averaged and temporal resolution was 16 to 18 ms.

Regional LV velocities were assessed by repeating this phase-contrast CMR sequence with a velocity encoding of 30 cm/s (tissue CMR) and the slice positioned during early diastole at 2/3 of the long-axis of the interventricular septum perpendicular to tissue movement as described previously (9) and encompassing the infarct area.

CMR image analysis

Offline analysis of short-axis images was performed using MASS analytical software (Medis, Leiden, The Netherlands). From the volume-versus-time curves, LV peak filling rate and time to peak filling rate were calculated (11). LV wall motion was assessed visually by two observers (B.P and H.J.L., 8 years and more than 15 years of experience in cardiac CMR, respectively) working together in consensus. Each segment was scored in a 17-segment model (12) using a 4-point scale (1 = normal wall motion, 2 = hypokinesia, 3 = akinesia, 4 = dyskinesia) (13). For each patient a wall motion score index was calculated as the sum of scores divided by the number of total segments.

The transmural extent of LGE was scored using the 17-segment model (13). Transmurality in each segment was visually assessed (B.P. and H.J.L. in consensus) on a 5-point scale: 0 = no hyperenhancement; 1 = 1–25% hyperenhanced; 2 = 26–50% hyperenhanced; 3 = 51–75% hyperenhanced, and 4 = 76–100% hyperenhanced (14). For each patient, a myocardial transmurality index was calculated as the sum of segmental scores divided by the number of segments. A transmurality index in the right coronary artery territory or inferior transmurality index was assessed separately as the sum of LGE score divided by 5 segments assigned to the right coronary artery (basal and mid inferior segments, basal posteroseptal, basal posterior and apical inferior segments). The extent of myocardial damage was traced and calculated using MASS analytical software package and expressed as percentage of total LV mass (Medis, Leiden, The Netherlands) (15).

From the transmitral flow curves, early (E) and late (A) LV filling velocity and E/A ratio was determined (9). Analysis of longitudinal myocardial tissue velocities was done offline by one author (B.P.). A standardized circular region of interest (ROI) of 20 pixels was placed at different locations in the LV myocardium (Fig. 1). The measured myocardial regions were: inferior (= infarcted); lateral and septal (= adjacent); and anterior (= remote). The ROI was traced on the modulus image, adjusted in each phase for cardiac motion and was transferred to the paired phase image, using the FLOW analytical software package (Medis, Leiden, The Netherlands). For each ROI, peak systolic velocity (Sa) and peak early diastolic velocity (Ea) was measured (Fig. 1). Regional velocities were measured in the same standardized ROI position in control subjects. In addition, average early diastolic longitudinal tissue velocity of infarcted, adjacent and remote myocardium or average Ea was calculated as a measure of global diastolic LV function.

Statistical analysis

Data are presented as mean ± SD. An unpaired two-tailed *t* test was used to compare parameters between patients and control subjects. Significance was set at $p < 0.05$. All statistical analyses were performed by using computer software (SPSS for Windows, version 12.0; SPSS, Chicago, Illinois, USA).

RESULTS

Fifteen consecutive patients (male/female: 12/3) were included. Maximal creatine kinase MB was 224 ± 147 ng/mL. CMR was performed at 6 ± 3 days (range 2–12 days) after acute myocardial infarction.

Fifteen control subjects (male/female: 10/5) were normal at clinical examination, without a history of cardiovascular disease and without any complaints. The age of patients (52 ± 5 years) and healthy subjects (48 ± 6 years) was matched ($p = 0.10$). Subjects were normotensive, patients with a mean blood pressure of 89 ± 8 mm Hg, and healthy subjects with a mean

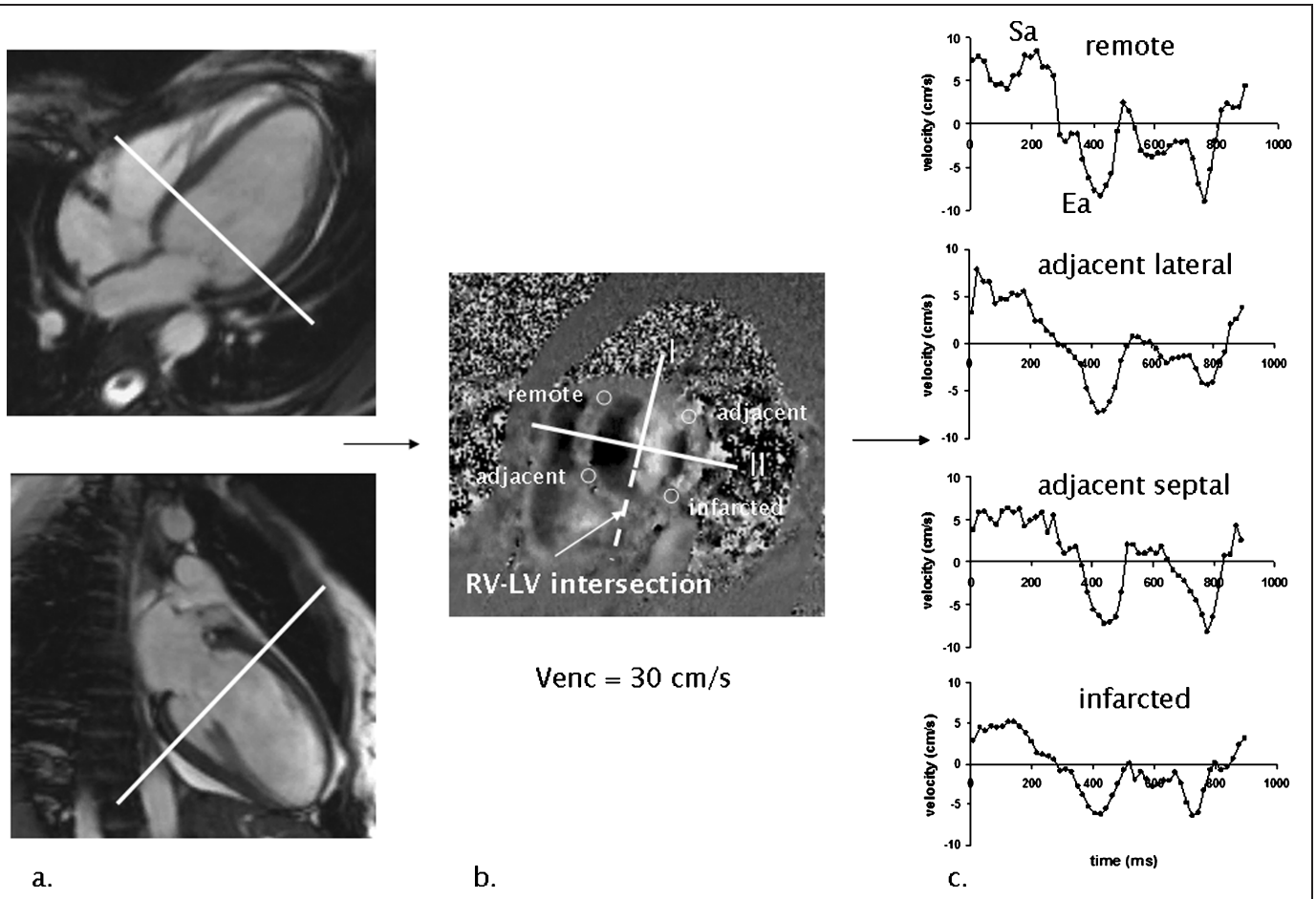

Figure 1. Assessment of myocardial velocities. (a) Velocity encoded MR imaging was performed with a velocity encoding of 30 cm/s, and the image slice positioned at 2/3 of the long axis, planned on early diastolic four- and two-chamber images, perpendicular to the interventricular septum. (b) Corresponding velocity encoded image. A standardized circular region of interest of 20 pixels (open circles) was placed at different locations around the circumference of the heart. A reference line (I) was traced through the center of the left ventricular (LV) cavity and the intersection of the posterior LV with the right ventricle (RV). Perpendicular on reference line (I) a second reference line (II) was positioned through the center of the LV cavity, dividing the LV myocardium into four regions. Regions of interest were placed in the center of the myocardium at the following locations: inferior (= infarcted); lateral (= adjacent); septal (= adjacent); and anterior (= remote) myocardium. (c) Velocity versus time curve in remote, adjacent lateral, adjacent septal and infarcted myocardium. Ea = peak early diastolic tissue velocity; LV = left ventricle; RV = right ventricle; Sa = peak systolic tissue velocity; Venc = velocity encoding.

blood pressure of 89 ± 13 mm Hg ($p > 0.99$). Heart rate was 67 ± 9 beats/min and 74 ± 15 beats/min ($p > 0.18$) for patients and control subjects, respectively.

Global left ventricular morphology and function

The culprit lesion was located in the proximal segment of the right coronary artery and after percutaneous intervention TIMI 3 flow was obtained. All patients had a transmural myocardial scar located in the LV inferior wall (including basal and mid segments of the inferior wall) (Table 1). Parameters of global LV systolic and diastolic function are shown in Table 1.

Global systolic LV function was depressed in the patients as compared to control subjects (ejection fraction: 46 ± 7% versus 57 ± 4%, $p = 0.000052$). Global diastolic LV function was impaired in patients as compared to controls, as indicated by impaired LV filling (peak filling rate: 301 ± 98 mL/s versus 430 ± 63 mL/s, $p = 0.00026$; time to peak filling rate: 238 ± 138 ms versus 152 ± 25 ms, $p = 0.032$), impaired average Ea (6.8 ± 1.7 cm/s versus 10.4 ± 1.5 cm/s, $p = 0.0000012$), and increased E/posteroseptal Ea (12.5 ± 4.0 versus 8.8 ± 5.3, $p = 0.00030$). E/A was not different between patients and controls.

Regional left ventricular function

Regional systolic velocities were reduced in patients as compared to controls, in the infarcted myocardium (inferior Sa: 5.5 ± 1.4 cm/s versus 7.5 ± 1.9 cm/s, $p = 0.002$), and adjacent myocardium (Table 2). Regional systolic velocities were preserved in the remote myocardium (remote Sa patients: 6.6 ± 2.0 cm/s versus remote Sa controls: 6.8 ± 1.4 cm/s, $p = 0.70$).

Regional diastolic LV velocities were impaired in patients in infarcted myocardium (Ea: 5.7 ± 2.0 cm/s versus 9.2 ± 2.8 cm/s,

Table 1. Scar Tissue, Global Systolic and Diastolic Left Ventricular Function

Parameter	Patients with Myocardial Infarction (n = 15)	Control Subjects (n = 15)	p value
Transmurality index	0.88 ± 0.3	0	0.0000006
Inferior transmurality index	3.0 ± 1	0	0.00000054
Scar tissue (%)	27 ± 8	0	0.00000005
Mass index (g/m^2)	81 ± 17	71 ± 11	0.080
End-diastolic volume/BSA (mL/m^2)	86 ± 20	72 ± 8	0.019
End-systolic volume/BSA (mL/m^2)	48 ± 6	31 ± 5	0.0019
Ejection fraction (%)	46 ± 7	57 ± 4	0.000052
Stroke index (mL/m^2)	39 ± 7	40 ± 4	0.48
Cardiac index (L/min/m^2)	2.8 ± 0.4	3.6 ± 0.8	0.0028
Wall motion score index	1.5 ± 0.3	1	0.0000089
Peak filling rate (mL/s)	301 ± 98	430 ± 63	0.00026
Time to peak filling rate (ms)	238 ± 138	152 ± 25	0.032
E (cm/s)	74 ± 21	76 ± 19	0.83
A (cm/s)	56 ± 14	51 ± 8	0.23
E/A	1.4 ± 0.4	1.5 ± 0.5	0.34
average Ea (cm/s)	6.8 ± 1.7	10.4 ± 1.5	0.0000012
E/posteroseptal Ea	12.5 ± 4.0	8.8 ± 5.3	0.00030

All values are mean ± SD. Parameters of scar tissue, global systolic and diastolic function were determined by magnetic resonance. A = peak mitral velocity at atrial contraction; BSA = body surface area; E = peak transmitral velocity in early diastole, Ea = early diastolic tissue velocity; average Ea represents average Ea of all regions (infarcted, adjacent and remote).

p = 0.00058), in adjacent myocardium (Table 2, lateral and septal), as well as in remote myocardium (Ea 7.2 ± 2.3 cm/s versus 10.2 ± 2.5 cm/s, p = 0.0026) as compared to control subjects (Table 2). Relative regional contribution to global diastolic LV function was expressed as percent difference of regional and average Ea. There was no difference in relative regional contribution to global diastolic LV function between patients and control subjects (Table 2).

DISCUSSION

In the present study, clinical application of tissue CMR demonstrated that early after reperfused transmural myocardial infarction of the inferior wall, regional longitudinal diastolic velocities were impaired in the infarcted, adjacent and remote tissue, whereas regional longitudinal systolic velocities were preserved in remote tissue.

Table 2. Regional Systolic and Diastolic Left Ventricular Function

Region	Sa (cm/s)		Ea (cm/s)		|Difference| between regional Ea and average Ea (%)	
	Patients	Controls	Patients	Controls	Patients	Controls
Inferior	5.5 ± 1.4	7.5 ± 1.9	5.7 ± 2.0	9.2 ± 2.8	−16 ± 18	−12 ± 22
	p = 0.002		p = 0.0006		p = 0.55	
Lateral	7.3 ± 1.7	8.8 ± 2.0	8.0 ± 3.2	12.6 ± 3.0	16 ± 31	20 ± 23
	p = 0.035		p = 0.00037		p = 0.65	
Septal	5.6 ± 1.2	8.5 ± 2.3	6.3 ± 1.8	9.8 ± 2.6	−5 ± 24	−5 ± 26
	p = 0.00033		p = 0.00027		p = 0.95	
Anterior	6.6 ± 2.0	6.8 ± 1.4	7.2 ± 2.3	10.2 ± 2.5	6 ± 17	−3 ± 16
	p = 0.70		p = 0.0026		p = 0.16	
Average	6.3 ± 1.0	8.6 ± 1.3	6.8 ± 1.7	10.4 ± 1.5	NA	NA
	p = 0.000014		p = 0.0000012			

All values are mean ± SD. E = peak transmitral velocity in early diastole; Ea = peak early diastolic tissue velocity; average Ea represents average Ea of all regions (infarcted, adjacent and remote); Sa = peak systolic tissue velocity. NA = non applicable.
Unpaired two-tailed Student *t* test control subjects versus patients.
Percent difference with average Ea was not significant between control subjects and patients.

Recently, quantification of regional myocardial function by measurement of myocardial velocities and by measurement of myocardial deformation or strain has been made possible. Myocardial velocities can be measured by tissue Doppler imaging and tissue CMR (7–9). Myocardial strain can be measured by a tissue Doppler imaging-based modality: strain Doppler echocardiography or strain rate imaging (16, 17) and by CMR tagging (18). The analysis of remote myocardial function has mainly been focused on anterior myocardial infarction (5, 19), demonstrating reduced longitudinal velocities using tissue Doppler imaging (20), impaired strains (3, 21) and strain rates (22, 23) in the remote myocardium using strain rate imaging and CMR tagging.

In this study, regional longitudinal function was assessed in the early phase after myocardial infarction of the inferior wall. For this purpose, longitudinal myocardial velocities were measured using tissue CMR.

In the presently studied patients with myocardial infarction of the inferior wall, regional longitudinal systolic LV velocities (Sa) were preserved in the remote myocardium. In myocardial infarction of the anterior wall however, tissue Doppler imaging data have shown reduced myocardial velocities (20), and CMR tagging data have shown impaired longitudinal LV shortening in the noninfarcted myocardium (3, 4). In the presently studied patients with myocardial infarction of the inferior wall, only longitudinal diastolic LV velocities (Ea) of both the adjacent and the remote myocardium were found impaired. Longitudinal ventricular motion is mainly determined by the oblique clockwise oriented myocardial fibers in the subendocardium (24, 25). The circumferentially wrapped cardiac fibers in the middle layer and the oblique anticlockwise oriented cardiac fibers in the subepicardial layer mainly contribute to circumferential and radial function (26) .During LV filling the ventricle expands radically and longitudinally. Diastolic dysfunction is known to be a sensitive and early marker of myocardial disease (22, 27). In contrast to the subepicardial myocardium, the function of the subendocardial myocardium is especially sensitive and prone to ischemia (28, 29). Therefore, impairment of longitudinal diastolic velocities in the adjacent and remote myocardium may be an early sign of myocardial dysfunction. Although myocardial infarction of the inferior wall exhibits a smaller functional loss as compared to similar sized myocardial infarction of the anterior wall, impact on the remote region may be apparent from regional diastolic dysfunction.

Regional myocardial velocities are, however, not only determined by the function of the segment under investigation but also by traction and tethering from other myocardial segments and by translational motion of the entire heart (16). Motion of the heart base is basically the resultant sum of longitudinal motion between apex and base of the heart. Therefore, reduction of velocities in these basal segments does not always mean reduction in function of these segments. In the presently studied patients, reduction of myocardial velocities in the remote region during diastole might represent myocardial tethering. Myocardial deformation (strain) and rate of deformation (strain rate) are less influenced by cardiac translation and motion due to tethering from other segments. Assessment of strain rate in future research by using CMR tagging may overcome the limitations of velocity measurement (17).

Within hours of cardiomyocyte injury, infarct expansion results in wall thinning and ventricular dilatation causing elevation of systolic and diastolic wall stress (30). Increased wall stress stimulates adaptive hypertrophy and alterations in both the infarcted and surrounding non-infarcted ventricular myocardium architecture in order to distribute wall stresses more evenly. Late remodeling (beyond 72 hours) is characterized by distortion of the LV shape and hypertrophy in the remote myocardium. Increased remodeling and strain alterations in the non-infarcted myocardium have been related to the extent of microvascular obstruction within infarcted regions (31). Therefore, both the tethering effect of the infarcted myocardium and regional alterations in wall stress (5, 31, 32), together with reduced coronary vasodilation (31) might explain reduction of function in the adjacent and remote myocardium. Due to smaller impact on global LV function in patients with myocardial infarction of the inferior wall, only moderate LV remodeling (5, 6) and therefore moderately increased LV end diastolic volumes were found in the present study.

Global diastolic dysfunction and impaired relaxation was apparent in patients from the functional LV volumes showing a reduced LV peak filling rate and a prolonged LV time to peak filling rate. Posteroseptal Ea and average Ea which represent the net velocity changes of the heart during early diastole in LV long-axis dimension relate to the rate of myocardial relaxation (33). The transmitral flow pattern, including E/A ratio (1.5 ± 0.5 versus 1.4 ± 0.4 in patients, $p > 0.05$), however, was similar between control subjects (34, 35) and patients. Since posteroseptal Ea and average Ea were found to be reduced, the "normal" transmitral flow pattern in patients was most likely pseudonormal (9, 33).

In addition, dysfunction of the early diastolic longitudinal LV motion of both infarcted and non-infarcted myocardium might have contributed to global LV diastolic dysfunction. There was a mild increase in the relative contribution of the remote region to global early diastolic long-axis velocity (= average Ea). The remote region is relatively small, and this contribution was not statistically significantly different. Therefore, relative segmental contribution to global diastolic LV function in patients was similar to the segmental distribution in control subjects. Since early diastolic long axis LV motion precedes transmitral flow and influences LV filling (36), depressed longitudinal LV motion of infarcted, adjacent and remote myocardium affects LV filling due to decreased elastic recoil and impaired diastolic suction (37).

CONCLUSIONS

Assessment of regional velocities after myocardial infarction is feasible using tissue CMR. Regional diastolic velocities early after inferior myocardial infarction are impaired in the infarcted, adjacent and remote tissue. However, regional systolic velocities are preserved in remote tissue. Therefore, impairment of regional diastolic velocities can be an early marker of dysfunction in remote tissue after inferior myocardial infarction.

REFERENCES

1. Moller JE, Sondergaard E, Poulsen SH, et al. Pseudonormal and restrictive filling patterns predict left ventricular dilation and cardiac death after a first myocardial infarction: a serial color M-mode Doppler echocardiographic study. J Am Coll Cardiol 2000;36:1841–6.
2. Nijland F, Kamp O, Karreman AJ, et al. Prognostic implications of restrictive left ventricular filling in acute myocardial infarction: a serial Doppler echocardiographic study. J Am Coll Cardiol 1997;30:1618–24.
3. Bogaert J, Bosmans H, Maes A, et al. Remote myocardial dysfunction after acute anterior myocardial infarction: impact of left ventricular shape on regional function: a magnetic resonance myocardial tagging study. J Am Coll Cardiol 2000;35:1525–34
4. Kramer CM, Rogers WJ, Theobald TM, et al. Remote noninfarcted region dysfunction soon after first anterior myocardial infarction. A magnetic resonance tagging study. Circulation 1996;94:660–6.
5. Marino PN, Kass DA, Becker LC, et al. Influence of site of regional ischemia on nonischemic thickening in anesthetized dogs. Am J Physiol 1989;256:H1417–25.
6. Goto T, Ohte N, Miyabe H, et al. Tl-201 washout rate in remote normal regions in patients with prior myocardial infarction and left ventricular remodeling. J Nucl Cardiol 2005;12:179–85.
7. Arai AE, Gaither CC 3rd, Epstein FH, Balaban RS, Wolff SD. Myocardial velocity gradient imaging by phase contrast MRI with application to regional function in myocardial ischemia. Magn Reson Med. 1999;42:98–109
8. Karwatowski SP, Chronos NA, Sinclaire H, Forbat SM, St John Sutton MG, Black C, Underwood SR, Pennell DJ. Effect of systemic sclerosis on left ventricular long-axis motion and left ventricular mass assessed by magnetic resonance. J Cardiovasc Magn Reson. 2000;2:109–17.
9. Paelinck BP, de Roos A, Bax JJ, et al. Feasibility of tissue magnetic resonance imaging: a pilot study in comparison with tissue Doppler imaging and invasive measurement. J Am Coll Cardiol 2005;45:1109–16.
10. Kellman P, Arai AE, McVeigh ER, et al. Phase-sensitive inversion recovery for detecting myocardial infarction using gadolinium-delayed hyperenhancement. Magn Reson Med 2002;47:372–83.
11. Fujita N, Hartiala J, O'Sullivan M, et al. Assessment of left ventricular diastolic function in dilated cardiomyopathy with cine magnetic resonance imaging: effect of angiotensin converting enzyme inhibitor, benazepril. Am Heart J 1993;125:171–8.
12. Cerqueira MD, Weissman NJ, Dilsizian V, et al; American Heart Association Writing Group on Myocardial Segmentation and Registration for Cardiac Imaging. Standardized myocardial segmentation and nomenclature for tomographic imaging of the heart: a statement for healthcare professionals from the Cardiac Imaging Committee of the Council on Clinical Cardiology of the American Heart Association. Circulation 2002;105:539–42.
13. Geleijnse ML, Fioretti PM, Roelandt JR. Methodology, feasibility, safety and diagnostic accuracy of dobutamine stress echocardiography. J Am Coll Cardiol 1997;30:595–606.
14. Wu E, Judd RM, Vargas JD, Klocke FJ, Bonow RO, Kim RJ. Visualisation of presence, location, and transmural extent of healed Q-wave and non-Q-wave myocardial infarction. Lancet 2001;357:21–8.
15. Schuijf JD, Kaandorp TA, Lamb HJ, et al. Quantification of myocardial infarct size and transmurality by contrast-enhanced magnetic resonance imaging in men. Am J Cardiol 2004;94:284–8.
16. Urheim S, Edvardsen T, Torp H, Angelsen B, Smiseth OA. Myocardial strain by Doppler echocardiography. Validation of a new method to quantify regional myocardial function. Circulation 2000;102:1158–64.
17. Smiseth OA, Ihlen H. Strain rate imaging. Why do we need it? J Am Coll Cardiol 2003;42:1584–86.
18. Axel L, Dougherty L. Heart wall motion: improved method of spatial modulation of magnetization for MR imaging. Radiology 1989;172:349–350.
19. Uren NG, Crake T, Lefroy DC, et al. Reduced coronary vasodilator function in infarcted and normal myocardium after myocardial infarction. N Engl J Med 1994;331:222–7.
20. Skulstad H, Urheim S, Edvardsen T, et al. Grading of myocardial dysfunction by tissue Doppler echocardiography: a comparison between velocity, displacement, and strain imaging in acute ischemia. J Am Coll Cardiol 2006;47:1672–82.
21. Kramer CM, Rogers WJ, Theobald TM, et al. Dissociation between changes in intramyocardial function and left ventricular volumes in the eight weeks after first anterior myocardial infarction. J Am Coll Cardiol 1997;30:1625–32.
22. Azevedo CF, Amado LC, Kraitchman DL, et al. Persistent diastolic dysfunction despite complete systolic functional recovery after reperfused acute myocardial infarction demonstrated by tagged magnetic resonance imaging. Eur Heart J 2004;25:1419–27.
23. Pislaru C, Bruce CJ, Anagnostopoulos PC, et al. Ultrasound strain imaging of altered myocardial stiffness. Stunned versus infarcted reperfused myocardium. Circulation 2004;109:2905–10.
24. Greenbaum RA, Ho SY, Gibson DG, et al. Left ventricular fibre architecture in man. Br Heart J 1981;45:248–63.
25. Chan J, Hanekom L, Wong C, Leano R, Cho GY, Marwick TH. Differentiation of subendocardial and transmural infarction using two-dimensional strain rate imaging to assess short-axis and long-axis myocardial function. J Am Coll Cardiol 2006;48:2026–33.
26. Rademakers FE, Rogers WJ, Guier WH, et al. Relation of regional cross-fiber shortening to wall thickening in the intact heart. Three-dimensional strain analysis by NMR tagging. Circulation 1994;89:1174–82.
27. Henein MY, Priestley K, Davarashvili T, et al. Early changes in left ventricular subendocardial function after successful coronary angioplasty. Br Heart J 1993;69:501–6.
28. Nesto RW, Kowalchuk GJ. The ischemic cascade: temporal sequence of hemodynamic, electrocardiographic and symptomatic expressions of ischemia. Am J Cardiol 1987;59:23C–30C.
29. Henein MY, Gibson DG. Long axis function in disease. Heart 1999;81:229–231.
30. Sutton MG, Sharpe N. Left ventricular remodeling after myocardial infarction. pathophysiology and therapy. Circulation 2000;101:2981–8.
31. Kramer CM, Lima JA, Reichek N, et al. Regional differences in function within noninfarcted myocardium during left ventricular remodeling. Circulation 1993;88:1279–88.
32. Epstein FH, Yang Z, Gilson WD, et al. MR tagging early after myocardial infarction in mice demonstrates contractile dysfunction in adjacent and remote regions. Magn Reson Med 2002;48:399–402.
33. Ommen SR, Nishimura RA, Appleton CP, et al. Clinical utilility of Doppler echocardiography and tissue Doppler imaging in the estimation of left ventricular filling pressures. A Comparative simultaneous Doppler-catheterization study. Circulation 2000;102:1788–94.
34. Klein AL, Burstow DJ, Tajik AJ, Zachariah PK, Bailey KR, Seward JB. Effects of age on left ventricular dimensions and filling dynamics in 117 normal persons. Mayo Clin Proc 1994;69:212–24.
35. Diamant M, Lamb HJ, Groeneveld Y, et al. Diastolic dysfunction is associated with altered myocardial metabolism in asymptomatic normotensive patients with well-controlled type 2 diabetes mellitus. J Am Coll Cardiol 2003;42:328–35.
36. Karwatowski SP, Brecker SJ, Yang GZ, et al. A comparison of left ventricular myocardial velocity in diastole measured by magnetic resonance and left ventricular filling measured by Doppler echocardiography. Eur Heart J 1996;17:795–802.
37. Rademakers FE, Buchalter MB, Rogers WJ, et al. Dissociation between left ventricular untwisting and filling. Accentuation by catecholamines. Circulation 1992;85:1572–81.