Quantitative systolic and diastolic transmyocardial velocity gradients assessed by M-mode colour Doppler tissue imaging as reliable indicators of regional left ventricular function after acute myocardial infarction


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Aims The aim of this study was to determine whether myocardial velocity gradients assessed by M-mode colour Doppler tissue imaging could be of clinical relevance and represent reliable indicators of regional left ventricular function after acute myocardial infarction.

Methods and Results Among 64 consecutive patients with a first acute myocardial infarction, in 50 who had a marked asynergy in the parasternal short-axis view at the mid-papillary muscle level, myocardial velocities and velocity gradients were assessed in the anteroseptum and posterior wall by M-mode Doppler tissue imaging. Similar measurements were obtained in 11 matched healthy volunteers who served as a control group.

In patients with anterior myocardial infarction, the peak myocardial velocity gradient in the anteroseptum was significantly lower when compared with controls (0.9 ± 0.5 vs 1.8 ± 1.2 s⁻¹ during systole and 1.4 ± 1.4 vs 4.9 ± 1.2 s⁻¹ during diastole, both P<0.01). Conversely, the peak systolic myocardial velocity gradient in the posterior wall was significantly higher than in controls (2.6 ± 1.2 vs 1.8 ± 1.2 s⁻¹, P<0.05).

In patients with inferior myocardial infarction, the peak myocardial velocity gradient in the posterior wall was significantly lower when compared with healthy subjects (0.9 ± 0.6 vs 1.8 ± 1.2 s⁻¹ during systole and 1.4 ± 1.4 vs 4.9 ± 1.2 s⁻¹ during diastole, both P<0.01). The peak systolic tissue velocity gradient in the anteroseptum was significantly higher than in controls (2.1 ± 1.0 vs 1.1 ± 0.7 s⁻¹, P<0.01).

Conclusion The present study indicates that myocardial velocity gradients assessed by M-mode Doppler tissue imaging are of clinical relevance for the characterization of ischaemic myocardial dysfunction after infarction and may provide quantitative assessment of segmental left ventricular function in this clinical setting.

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Key Words: Echocardiography, Doppler, velocity, imaging, myocardium, myocardial infarction.

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Introduction

Assessment of left ventricular wall motion is one of the most important procedures in evaluating left ventricular function. Regional wall motion is closely related to the status of the cardiac muscle in these local areas[1-6]. However, it is still difficult to evaluate wall motion quantitatively in real time by conventional techniques[7-14]. To date, techniques allowing the assessment of left ventricular function have focused on the analysis of endocardial excursion[15-18]. More recently, automatic myocardial edge detection techniques have authorized the analysis of wall thickening as an indicator of regional left ventricular function[19-21].
Doppler tissue imaging is a recent technique, exhibiting the well-known advantages of diagnostic ultrasound, and able to produce images of the velocity of tissue motion within the myocardium\textsuperscript{[22]}. Originally, Doppler tissue imaging was developed as a qualitative aid for left ventricular wall motion analysis. It has been demonstrated by a series of in vitro studies using tissue-mimicking phantoms that qualitative assessment of myocardial velocity and acceleration derived from Doppler tissue imaging is reliable\textsuperscript{[23,24]}. More recently, investigations using Doppler myocardial imaging has concentrated on the quantitation of regional myocardial velocities\textsuperscript{[24,25]}. The in vitro validation of velocities has been described\textsuperscript{[24]}. There is a close correlation in healthy subjects between endocardial velocities derived from Doppler tissue imaging and those derived using digitized analysis of conventional M-mode echocardiography\textsuperscript{[26,27]}. To date, only a few studies have allowed accurate quantification of regional myocardial wall motion. In these studies, a transmural velocity gradient was noted in normal subjects whereby velocities increased from the epicardium through the myocardium to the endocardium\textsuperscript{[25–27]}. Furthermore, it has been postulated that the systolic myocardial velocity gradient could be a new indicator of regional left ventricular contraction in the setting of dilated cardiomyopathy or prior myocardial infarction\textsuperscript{[25]}. Because M-mode colour Doppler myocardial imaging affords greater temporal resolution and frame rates than two-dimensional imaging\textsuperscript{[28]}, M-mode Doppler tissue imaging may be a very accurate method for quantitation of myocardial velocity gradients. Conversely, the quantitative assessment of myocardial wall motion velocity by M-mode colour Doppler tissue imaging is limited to the anteroseptum and the posterior wall for which the direction of wall motion occurs parallel to the Doppler beam. Finally, whether the quantitative assessment of systolic and diastolic myocardial velocity gradients by M-mode colour Doppler tissue imaging could be of clinical relevance in a large population of patients who suffered a recent myocardial infarction has not been specifically addressed.

The present study was designed to determine whether systolic and diastolic myocardial velocity gradients assessed by quantitative M-mode colour Doppler tissue imaging could represent reliable indicators of regional left ventricular function in the setting of acute myocardial infarction, by comparing transmural velocity gradients obtained in the corresponding walls in a large population of recently infarcted patients and in matched healthy volunteers.

**Methods**

**Doppler tissue imaging system**

We used conventional ultrasound equipment (Acuson 128 XP/10, Mountain View, CA, U.S.A.) with a phased array transducer 2·5 to 4 MHz and software modifications which allowed the display of regional velocities within the myocardium by M-mode colour Doppler tissue imaging. The concept and technical aspects of the imaging system used in this study have been described in detail elsewhere\textsuperscript{[23,24,29]}. To permit the acquisition of velocity information, the sensitivity of the scanner had to be reduced to allow the tissue echoes to pass through the clutter filter and to remove the blood signals, i.e. to accommodate high echo-amplitude and low Doppler velocity signals. The Doppler velocity range was also reduced to correspond to the known velocities of the ventricular wall. These adjustments enabled Doppler velocity mapping of myocardial motion in the range of $-30$ to $+30$ cm $\cdot$ s$^{-1}$. The narrowest velocity range which could be encoded over the full colour spectrum was $-3$·0 to $3$·0 cm $\cdot$ s$^{-1}$, allowing a very good velocity resolution of 0·2 cm $\cdot$ s$^{-1}$. Pulsed repetition frequency was 3–6 KHz and the Doppler sampling rate in the M-mode could reach 100–110 Hz.

**Image acquisition**

An M-mode study of the parasternal short-axis view of the left ventricle at the mid-portion of the papillary muscle level was performed in healthy subjects and in infarct patients. The colour Doppler box was placed over the region of interest, i.e. anteroseptal and posterior walls, of the standard grey-scale image. All scanner settings were kept constant throughout the study except for grey-scale receive gain, Doppler receive gain, Doppler velocity range and image magnification. Grey-scale receive gain was set to achieve an acceptable image of the myocardium allowing a good detection of epicardial and endocardial boundaries. This was followed by switching off the B-mode transmit power and gain and the colour Doppler gain was adjusted to obtain maximal in-filling of the myocardium without significant noise interference, i.e. when noticeable colour degradation occurs. The Doppler velocity scale was reduced to the lowest setting at which aliasing did not occur. Image magnification was increased as much as possible while allowing the myocardial wall to stay within the frame for the entire cardiac cycle. Echocardiographic images were recorded on S-VHS videotape for off-line digitization. The format of the captured images was 512 horizontal pixels by 288 vertical pixels with 24 bits per pixel representing red, green and blue components of the image. The files were transferred to a PC compatible computer for off-line processing.

**M-mode determination of wall motion velocity**

Peak endocardial and epicardial excursion velocities of the left ventricular posterior and anteroseptal walls were determined in all patients by analysing the Doppler shift
from the colour images using a dedicated software environment for vision applications (Visilog, developed by Noesis, France), which made it possible to convert the analog representation of colours into velocity estimates using the colour values obtained from the colour scale displayed on colour Doppler images. Care was taken to achieve good detection of both endocardial and epicardial boundaries in 2D-mode tomograms. In colour Doppler images, we included all of the myocardium between the two boundaries. Peak motion velocity at each endocardial and epicardial site was then determined, in the same vertical scan-line of M-mode colour tomograms using the average velocity of a single-size (1 mm²) manually selected region of interest, exhibiting maximal colour in-filling (Fig. 1). It is now well established that myocardial velocities during systole and diastole are not constant, with a single peak during systole and two velocity peaks during diastole. Because diastolic myocardial velocities are higher during early diastole in healthy subjects, peak myocardial velocities and velocity gradients assessed during diastole in the current study always referred to early diastole. Thus, the diastolic myocardial velocity gradient was always measured in the same part of the cardiac cycle for the overall population. The peak myocardial velocity gradient assessed in both walls was defined by: MVG=(Vendo-Vepi)/T, with MVG: myocardial velocity gradient, Vendo: peak endocardial excursion velocity, Vepi: peak epicardial excursion velocity, T: wall thickness.

**Study group**

During the study period, 64 consecutive patients with the diagnosis of a first acute myocardial infarction with normal sinus rhythm and no bundle branch block were screened to participate in the study (34 with anterior and 30 with inferior acute myocardial infarction). The diagnosis of acute myocardial infarction had been confirmed by electrocardiographic features, a rise in creatine kinase MB enzymes and a marked segmental asynergy at conventional echocardiography. To be included in the study, wall motion abnormalities of the anteroseptum or the posterior wall had to be easily imaged and well seen by echocardiography in the short-axis plane at the mid-papillary muscle level and written informed consent had to be obtained from each patient. Echocardiographic images were recorded within the first week after the onset of myocardial infarction. The control group comprised 11 matched healthy volunteers who had normal conventional echocardiographic findings and no history of prior cardiomyopathy, angina, arrhythmias, systemic hypertension or diabetes mellitus.

**Statistical analysis**

Data are shown as mean value ± standard deviation. We used a paired Student t-test to analyse data within a group and an unpaired t-test to analyse data in different groups. Data were considered significant at a P value <0.05.

The reproducibility for the measurements of peak myocardial velocities was assessed in six randomly selected normal volunteers by two independent investigators. Results are expressed as the mean ± SD difference, and correlations were calculated by the linear regression method.

**Results**

**Study population**

Among 64 consecutive screened patients, the study population comprised 26 patients with anterior acute myocardial infarction (20 men, six women, mean age 63 ± 13 years) and 24 patients with inferior acute myocardial infarction (19 men, five women, mean age 61 ± 14 years), who had a marked asynergy at the mid-level of the left ventricle, representing 78% of the screened population. The delay between the onset of myocardial infarction and Doppler tissue images acquisition was 3 ± 2 days. Eight patients with an anterior infarct could not participate the study because of poor acoustic windows in two patients and because wall motion abnormalities were not sufficiently visible at the mid-papillary muscle level in six patients; similarly, six patients with an inferior infarct were excluded because of poor acoustic windows in two patients and because there was no evidence of wall motion abnormalities at the mid-level of the left ventricle in the short-axis-view in four patients. Thus, quantitative analysis of wall motion velocity was available in 50 patients with acute myocardial infarction. Out of these, 46 had a Q wave and four a non-Q wave infarct. Doppler tissue images obtained in healthy subjects were all suitable for analysis (n=11, eight men, three women, mean age 53 ± 7 years).

**Quantitative assessment of myocardial velocity and myocardial velocity gradient in healthy subjects**

In controls, there was a regional heterogeneity in myocardial wall motion, with peak velocities that were greater in the posterior wall than in the anteroseptum (51 ± 12 vs 38 ± 5 mm.s⁻¹ for peak systolic endocardial excursion velocity, P=0.008; 137 ± 20 vs 56 ± 7 mm.s⁻¹ for peak diastolic endocardial excursion velocity, P<0.0001). The peak myocardial velocity gradient was lower in the anteroseptum when compared with that obtained in the posterior wall (systolic velocity gradient 1.1 ± 0.7 vs 1.8 ± 1.2 s⁻¹, P=0.05; diastolic velocity gradient 2.0 ± 0.5 vs 4.9 ± 1.2 s⁻¹, P<0.0001). Velocities were also significantly lower during systole compared with diastole in both walls (for peak endocardial excursion velocity in the anteroseptum 38 ± 5
Quantitative assessment of myocardial velocity and myocardial velocity gradient in patients with acute myocardial infarction

Peak myocardial velocities and myocardial velocity gradients are summarized in Table 1. Figure 2 shows examples of colour M-mode Doppler tissue images of the anteroseptum and the posterior wall in infarct patients and in healthy subjects.

In patients with anterior infarcts, peak epicardial and endocardial excursion velocities in the anteroseptal wall during systole and diastole were significantly lower when compared with controls. In this wall, as shown in Fig. 3, peak myocardial velocity gradients were dramatically decreased and significantly lower than in the control group (systolic velocity gradient 0.0 ± 0.5 vs 1.1 ± 0.7 s⁻¹, P < 0.001; diastolic velocity gradient 0.3 ± 0.6 vs 2.0 ± 0.5 s⁻¹, P < 0.0001). In the posterior wall, although peak systolic velocities were not significantly different from controls, the peak systolic myocardial velocity gradient was significantly higher (2.6 ± 1.2 vs 1.8 ± 1.2 s⁻¹, P < 0.05) (Fig. 3).

In patients with inferior infarcts, the peak epicardial and endocardial excursion velocities in the posterior wall were significantly lower when compared with those obtained in healthy subjects. In this wall, peak myocardial velocity gradients were significantly lower when compared with controls, particularly during diastole (1.4 ± 1.4 vs 4.9 ± 1.2 s⁻¹, P < 0.001) (Fig. 3). In the anteroseptum, although peak velocities were not significantly different from controls, the peak systolic myocardial velocity gradient was significantly higher (2.1 ± 1.0 vs 1.1 ± 0.7 s⁻¹, P = 0.001) (Fig. 3).

Out of the 50 patients who experienced an acute infarct, seven had dyskinetic segments as assessed by two-dimensional echocardiography (five anterior infarcts and two inferior). In each case, dyskinesia was accurately detected by colour-coded velocities, derived from M-mode DTI. Very interestingly, the transmyocardial velocity gradient was dramatically decreased in these patients and negative in five (for the anterior wall −0.6 ± 0.4 s⁻¹ during systole and −0.3 ± 0.4 s⁻¹ during diastole; for the posterior wall −0.2 ± 0.6 s⁻¹ and 0.0 ± 0.7 s⁻¹, respectively).

Reproducibility of results in healthy subjects

In healthy subjects, there were reproducible results between two independent investigators as regards peak systolic epicardial and endocardial excursion velocities in the anteroseptum and in the posterior wall. The mean difference between the two analyses were 56 ± 7 mm . s⁻¹, and in the posterior wall 51 ± 12 vs 137 ± 20 mm . s⁻¹, both P < 0.01).
Table 1  Peak myocardial velocity and myocardial velocity gradient assessed by M-mode colour Doppler tissue imaging in the anteroseptum and the posterior wall

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<tr>
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<th>Control (n=11)</th>
<th>Anterior infarct (n=26)</th>
<th>Inferior infarct (n=24)</th>
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<td>Peak systolic velocity and gradient</td>
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<td>V epi</td>
<td>25±5</td>
<td>28±8</td>
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<td>V endo</td>
<td>38±5§</td>
<td>51±12§</td>
<td>9±11†</td>
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<td>Velocity gradient (MVG)</td>
<td>1·1±0·7</td>
<td>1·8±1·2§</td>
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<td>Peak diastolic velocity and gradient</td>
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<td>V epi</td>
<td>29±6</td>
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<td>V endo</td>
<td>56±7</td>
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<td>Velocity gradient (MVG)</td>
<td>2·0±0·5</td>
<td>4·9±1·2§</td>
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In the anteroseptum, myocardial wall motion velocities and myocardial velocity gradients were consistently lower in patients with anterior infarction when compared with the corresponding wall in the control group. In the posterior wall, myocardial wall motion velocities and myocardial velocity gradients were consistently lower in patients with recent inferior infarction when compared with the corresponding wall in the control group. Data in patients with acute myocardial infarction are compared with controls. *P<0·01, †P<0·001, ‡P<0·05 vs control group, §P<0·01 vs diastole in healthy subjects, ¶P<0·01 vs anteroseptum in healthy subjects, ††P<0·05 vs anteroseptum in healthy subjects.

V epi=peak epicardial excursion velocity, V endo=peak endocardial excursion velocity, MVG=peak myocardial velocity gradient, AS=anteroseptum, PW=posterior wall.

0·6±1·0 (2·4%), 0·8±0·7 (2·2%), 0·8±0·3 (2·9%) and 0·9±0·2 mm·s⁻¹ (1·8%) for peak systolic epicardial velocity in the anteroseptum, peak systolic endocardial velocity in the anteroseptum, peak systolic epicardial velocity in the posterior wall, and peak systolic endocardial velocity in the posterior wall, respectively. For each variable, the two serial measurements were correlated: r=0·95 (P=0·003), r=0·96 (P=0·001), r=0·96 (P=0·001) and r=0·97 (P=0·001) respectively.

Discussion

The present study shows that both systolic and diastolic transmyocardial velocity gradients assessed by M-mode Doppler tissue imaging were consistently lower in the recently infarcted myocardial walls compared with the same segments in the control group. Uematsu et al. showed, in a small population of patients with old myocardial infarction, that the systolic myocardial velocity gradient, as assessed by two-dimensional Doppler tissue imaging, was significantly lower in the infarct region. These results suggest that systolic and diastolic myocardial velocity gradients, as measured by M-mode colour Doppler myocardial imaging, have potential for quantitative assessment of segmental left ventricular function in the infarct zone. Moreover, M-mode Doppler tissue imaging appears as a technique of great value for identifying and quantifying compensatory kinetics of the opposite wall after acute myocardial infarction, as suggested by an increase in the systolic myocardial velocity gradient in the remote myocardial region. In the current study, this promising imaging technique has also allowed the accurate detection of dyskinetic segments and the calculation of myocardial velocity gradients that were dramatically decreased and usually negative in these segments.

Our data confirmed that systolic and diastolic myocardial regional velocities, as assessed by colour M-mode Doppler tissue imaging, were significantly reduced in the infarct region when compared with the corresponding walls in healthy subjects. Although this point remains controversial, the presence of preserved myocardial velocity signals within akinetic segments was observed in the present study, showing that Doppler tissue imaging is an accurate tool able to detect extremely low velocity. In agreement with Miyatake et al., our results confirmed that measurement variations derived from colour Doppler myocardium imaging were very low (<3% in our study), showing that assessment of myocardial velocity was reliable and reproducible in the clinical setting.

Very interestingly, our study demonstrates that quantitative assessment of myocardial velocities and gradients by the M-mode Doppler tissue imaging technique in the parasternal short-axis view is feasible and of clinical relevance for the quantitative echocardiographic evaluation of regional left ventricular function in patients with acute myocardial infarction. This echocardiographic view offers the simultaneous assessment of the anteroseptal and the posterior walls, which are the most commonly involved in acute myocardial ischaemic dysfunction in the setting of myocardial infarction. In the present study, a marked asynery at the mid-papillary muscle level could be clearly identified in 78% of the screened infarct patients. Furthermore, the effect of the Doppler angle of incidence could be neglected in this echocardiographic view for both walls studied. The rationale for choosing M-mode
Doppler tissue imaging relies on the fact that, analogous to colour flow Doppler M-mode imaging, M-mode Doppler myocardial imaging affords greater temporal resolution and signal-to-noise ratio in a single spatial dimension than two-dimensional colour Doppler imaging. Conversely, two-dimensional colour Doppler
Figure 2  (c)–(d).

Figure 2  Colour M-mode Doppler tissue tomograms of the anteroseptal (a) and (b) and the posterior wall (c) and (d) obtained in a healthy subject (a) and (c), and in patients with acute myocardial infarction (anterior (b) and inferior infarct (d)), using the same Doppler colour scale in the corresponding wall. We observed lower peak systolic and diastolic velocities in the recently infarcted wall when compared with those obtained in the same segment in controls. Peak systolic and diastolic myocardial velocity gradients were also markedly reduced.
tissue imaging was limited on the current equipment by frame rates of 20 to 40 Hz. In a previous study, we have shown that the lower signal-to-noise ratio could impact on velocity estimators of the Doppler tissue imaging system and lead to underestimated myocardial velocity estimates in humans[29]. Thus, we suggest that quantitative assessment of regional left ventricular contraction abnormalities may be better studied on the current echocardiographs by the myocardial velocity gradient derived from quantitative M-mode colour Doppler tissue imaging than by any other means. In the present study, the myocardial velocity gradient appeared as a quantitative and reliable indicator of regional left ventricular function in patients with acute myocardial infarction. Nevertheless, further studies regarding the velocity distribution internal to the wall are required and could provide new insights into the pathophysiology of the ischemic dysfunction within the myocardial wall thickness.

**Myocardial velocities assessed by quantitative M-mode colour Doppler tissue imaging in healthy subjects**

As previously reported[26,27], our results in healthy subjects led to the same observation of regional heterogeneity in myocardial wall velocities. In subjects with no regional left ventricular wall motion abnormalities, previous studies and our results have consistently demonstrated systolic and diastolic velocities that are greater in the posterior wall than in the anteroseptum[26,27] and a higher myocardial velocity gradient in this wall when compared with that obtained in the anteroseptum at the same phase of the cardiac cycle. In agreement with these studies, we reported that velocities were lower during systole compared with diastole.

**Study limitations**

According to the technique described in this study, absolute quantitation of regional myocardial velocities and gradients was limited to the posterior and anteroseptal walls, for which systolic and diastolic motion occurred parallel to the direction of Doppler interrogation. Uematsu et al. described a method for the assessment of myocardial velocities that is theoretically independent of the Doppler angle of incidence[25]. However, this algorithm could limit the ability to resolve spatial velocity differences within myocardial layers. Furthermore, since cardiac rotation during contraction appears to be very heterogeneous in the different walls and during the cycle[30–36], an algorithm homogeneously applied to correct for angle dependency in the different segments could lead to inappropriate reconstructed velocities by the Doppler tissue imaging technique. Although this point remains a major limitation of the imaging technique we described in this work, our study showed the good feasibility and reliability of M-mode Doppler myocardial imaging for quantitative assessment of wall motion in a large population of consecutive patients who recently suffered either a first anterior or inferior infarct.

Quantitative assessment of regional left ventricular wall motion is still difficult because of the effects of the parallel motion of the whole heart[23,25]. However, in the parasternal short-axis view M-mode colour Doppler tissue imaging was able to minimize these technical limitations, firstly because rotational velocity components are almost perpendicular to the Doppler beam in these walls[30], and thus could be neglected. Secondly, the upward shift of the whole heart during contraction is not one of the most important components of cardiac motion during cycle[32–40]. Conversely, the longitudinal translation of the whole heart still remains a drawback of this technique, particularly for absolute quantitation of myocardial velocities within myocardial layers. However, as far as myocardial velocity gradient is concerned, Uematsu et al. recently showed, in a population of patients with uncomplicated atrial septal defect and exaggerated translation of the left ventricle, that myocardial velocity gradient measurement was independent from the translation of the heart[41].

As widely accepted, the tissue velocity gradient was analysed under the assumption that wall velocity varies linearly with depth. However, non-linear contraction...
through the myocardium is suggested by the differing behaviour of longitudinal and circumferential fibres[45].

In some scans, it was impossible to achieve complete myocardial colour in-filling, particularly when cardiac boundaries were blurred or in the case of poor echogenity, corresponding to a poor signal-to-noise ratio of the colour Doppler images. More recent echocardiographs will probably be able to minimize this drawback by providing greater frame rates and higher signal-to-noise ratios of colour Doppler myocardial images.

Although quantitative assessment of regional myocardial velocities is feasible with current echocardiographs, it is limited to extracting the instantaneous velocity at the sampled site selected by the cursor on the colour Doppler frame. In the present study, we developed a dedicated computer software and a second off-line processing algorithm to extract numeric velocities from a recorded colour region.

Since current magnetic resonance imaging techniques for the calculation of myocardial velocities are not widely accepted, the present study does not contain direct validation methodology for the myocardial velocity gradient calculations because of the unavailability of another independent standard of reference for velocity gradient in humans. Future work designed to compare myocardial velocities assessed by Doppler tissue imaging and magnetic resonance imaging techniques is warranted.

**Future clinical applications in myocardial ischaemia**

Reversible ischaemia, which results in the development of wall motion abnormalities only after stressing the myocardium, should be identified and quantified more reliably with less operator dependence, thus overcoming one of the major limitations of stress echocardiography[43]. This would require the development of real-time algorithms to quantitate regional wall motion abnormalities, providing an objective index of myocardial ischaemia during echocardiographic stress testing. Similarly, because this technique provides instantaneous quantitation of wall motion, quantitation of regional left ventricular contraction should be useful when assessing myocardial viability by intra-individual comparisons during dobutamine stress echocardiography. However, the method is to date limited in its current quantitative applications in the off-line accessibility of quantitative velocity data. Thus, further studies are required to develop an on-line, high speed and large quantity image acquisition and storage system. With further development, Doppler tissue imaging might allow a complementary assessment of regional myocardial perfusion after intravenous injection of ultrasound contrast agents[23]. Based on the previous observation that ischaemia develops through the wall thickness as a ‘wave-front’[44,45], Doppler tissue imaging could be of great value for velocity distribution internal to the wall. The potential of identifying subendocardial impairment due to non-transmural ischaemia that is not detectable by two-dimensional echocardiography could add important information during provocative tests. It has also been noted that several changes in myocardial layer velocities derived from Doppler tissue imaging could represent a mean for the non-invasive assessment of vessel patency early after the onset of acute myocardial infarction[46,47]. If successful, the non-invasive prediction of vessel patency after thrombolytic therapy, based on serial assessment of regional systolic and diastolic myocardial velocities, could impact on the need for early invasive testing and rescue angioplasty in patients treated with thrombolytics during acute myocardial infarction.

**Conclusion**

The present study clearly shows that the assessment of systolic and diastolic myocardial velocity gradients by M-mode colour Doppler tissue imaging is of clinical relevance in patients who recently suffered an acute myocardial infarction. Both systolic and diastolic trans-myocardial velocity gradients have the potential to represent quantitative and reliable indicators of regional left ventricular function in the asynergic zone after myocardial infarction. Compensatory kinetics of the opposite wall can be easily identified and are associated with an increase in systolic myocardial velocity gradient. Although several problems remain to be resolved, these data strongly suggest that quantitative M-mode colour Doppler tissue imaging is a useful technique for the characterization of myocardial wall motion and function after acute myocardial infarction. This non-invasive technique can give unique information in the study of recently infarcted myocardium based on the measurements of wall velocity from epicardium to endocardium and myocardial velocity gradient. Further studies regarding the velocity distribution internal to the wall are required to provide new insights into the pathophysiology of myocardial ischaemic dysfunction.

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