Assessment of Dyssynchronous Wall Motion During Acute Myocardial Ischemia Using Velocity Vector Imaging

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OBJECTIVES The purpose of this study was to investigate the diagnostic value of velocity vector imaging (VVI) for detecting acute myocardial ischemia and whether VVI can accurately demonstrate the spatial extent of ischemic risk area.

BACKGROUND Using a tracking algorithm, VVI can display velocity vectors of regional wall motion overlaid onto the B-mode image and allows the quantitative assessment of myocardial mechanics. However, its efficacy for diagnosing myocardial ischemia has not been evaluated.

METHODS In 18 dogs with flow-limiting stenosis and/or total occlusion of the coronary artery, peak systolic radial velocity ($V_{SYS}$), radial velocity at mitral valve opening ($V_{MVO}$), peak systolic radial strain, and the percent change in wall thickening (%WT) were measured in the normal and risk areas and compared to those at baseline. Sensitivity and specificity for detecting the stenosis and occlusion were analyzed in each parameter. The area of inward velocity vectors at mitral valve opening (MVO) detected by VVI was compared to the risk area derived from real-time myocardial contrast echocardiography (MCE). Twelve image clips were randomly selected from the baseline, stenosis, and occlusions to determine the intra- and inter-observer agreement for the VVI parameters.

RESULTS The left circumflex coronary flow was reduced by 44.3 ± 9.0% during stenosis and completely interrupted during occlusion. During coronary artery occlusion, inward motion at MVO was observed in the risk area. Percent WT, peak systolic radial strain, $V_{SYS}$, and $V_{MVO}$ changed significantly from values at baseline. During stenosis, %WT, peak systolic radial strain, and $V_{SYS}$ did not differ from those at baseline; however, $V_{MVO}$ was significantly increased ($-0.12 \pm 0.60$ cm/s vs. $-0.96 \pm 0.55$ cm/s, $p = 0.015$). Sensitivity and specificity of $V_{MVO}$ for detecting ischemia were superior to those of other parameters. The spatial extent of inward velocity vectors at MVO correlated well with that of the risk area derived from MCE ($r = 0.74$, $p < 0.001$ with a linear regression).

CONCLUSIONS The assessment of VVI at MVO permits easy detection of dyssynchronous wall motion during acute myocardial ischemia that cannot be diagnosed by conventional measurement of systolic wall thickness. The spatial extent of inward motion at MVO suggests the size of the risk area. (J Am Coll Cardiol Img 2008;1:210–20) © 2008 by the American College of Cardiology Foundation
The assessment of regional wall motion abnormalities in left ventricular (LV) myocardium is necessary for diagnosing ischemic heart disease. In particular, post-systolic thickening or shortening, which is defined as myocardial contraction after aortic valve closure (AVC), has been noted as a highly sensitive marker of myocardial ischemia. The analysis of regional myocardial velocity, strain rate, or strain assessed using the tissue Doppler technique permits sensitive detection of post-systolic thickening and improves the accuracy of diagnosis of ischemic heart disease (1–4). However, the angle dependency of the Doppler technique frequently hampers the evaluation of regional wall motion abnormalities, including post-systolic thickening, in ischemic myocardium (5–7).

B-mode tissue tracking is a promising method for evaluating regional wall motion without angle dependency (8–10). Using a novel feature-tracking algorithm, velocity vector imaging (VVI) can display velocity vectors of regional wall motion overlaid onto the B-mode image and allow the quantitative assessment of LV myocardial mechanics (11,12). We have already reported that abnormal inward wall motion, which is presumed to be due to post-systolic thickening, can be detected in the latter half of isovolumic relaxation during myocardial ischemia using VVI (13). However, its efficacy for diagnosing myocardial ischemia has not been elucidated. Because dyssynchronous wall motion of LV myocardium can be easily visualized using velocity vectors, we hypothesized that VVI would allow sensitive detection of dyssynchrony induced by post-systolic thickening during ischemia and be able to demonstrate the accurate spatial extent of the ischemic risk area without angle dependency.

In this study, we investigated the diagnostic value of VVI for detecting the critical state of acute myocardial ischemia by comparing it with the conventional measurement of systolic wall thickening in anesthetized dogs. We also evaluated whether the spatial extent of post-systolic inward motion detected by VVI corresponds with that of ischemic myocardium indicated by myocardial contrast echocardiography (MCE).

Methods

Animal preparation. All animal studies were performed in accordance with guidelines for the care and use of laboratory animals at our institution. Eighteen open-chest dogs were used in this study. Dogs (weighing 14.1 ± 0.6 kg) were anesthetized using intravenous pentobarbital sodium (35 mg/kg), intubated, and ventilated with room air using a respirator pump. An 18-gauge peripheral intravenous catheter positioned in the foreleg was used for administration of fluids, drugs, and contrast microbubbles during MCE. Anesthesia with pentobarbital sodium was maintained throughout the experiment (6 to 8 mg/kg/h). A 5-F catheter was placed in the ascending aorta to monitor blood pressure, and the electrocardiogram was monitored continuously.

Dogs were placed in the right recumbent position. A left lateral thoracotomy was performed, and the heart was suspended in a pericardial cradle. The proximal portion of the left circumflex artery (LCx) and/or left anterior descending artery (LAD) was dissected free from surrounding tissues, and a vascular occluder was placed to create a flow-limiting stenosis or total occlusion. A perivascular ultrasonic flow probe was placed at the distal site of the occluder and connected to a digital flowmeter (Transonic Systems, Ithaca, New York). Flow-limiting stenosis was set to be approximately half of the baseline flow, in which systolic wall thickening is relatively preserved (14).

Echocardiography. VVI. Echocardiography was performed using a Sequoia ultrasound system (Siemens Medical Solutions, Mountain View, California). The LV short-axis view at the papillary muscle level was visualized using a water bath as a standoff. The position of the ultrasound transducer for the short-axis view was fixed with a mechanical arm. Two-dimensional images (transmitting and receiving frequencies 2.0 and 4.0 MHz, respectively) for regional wall motion assessment were captured over 3 consecutive cardiac cycles. The frame rate was set at 80 to 84 frames/s. For the detailed evaluation of regional wall motion, the timing of mitral valve opening (MVO) was determined by measurement of mitral inflow in the apical 4-chamber view by pulse Doppler (2.0 MHz) and that of AVC was assessed from the aortic component of the second heart sound derived from the phonocardiogram, which was simultaneously displayed with Doppler data. When the apical view was scanned, the transducer was removed from the mechanical arm and held manually. A microphone for the phonocardiogram was placed directly on the base of the aorta. Data were digitally stored on magneto-optical disks for subsequent off-line analysis.

High frame rate acoustic capture B-mode data were analyzed using off-line software (Syngo Ve-
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and radial velocity at MVO (VMVO) as a parameter of motion. As a conventional measurement of reperfused area, we measured peak systolic radial velocity vectors of the center of the risk area derived from real-time MCE and the opposite normal epicardial borders was calculated. Zero strain was previously, and radial strain between the endocardial and epicardial borders was calculated. This determined the motion of the tissue along the tissue. The velocity vector of each point in the border was given by the calculated displacements divided by the time interval between frames. Spatial coherence in the tracked border was done by applying a 3-point median filter and a 3-point Gaussian filter. Sixty velocity vectors were displayed on each image in our setting. A point of reference was placed by the user at the center of the LV cavity to determine the radial myocardial velocities.

For measuring transmural radial strain, we manually placed the same number of epicardial points as defined in the endocardium. The epicardial border was tracked using the same algorithm as mentioned previously, and radial strain between the endocardial and epicardial borders was calculated. Zero strain was set at the peak R-wave in electrocardiography.

Time-velocity curves were analyzed from 5 velocity vectors of the center of the risk area derived from real-time MCE and the opposite normal perfused area. We measured peak systolic radial velocity ($V_{SYS}$) as a parameter of systolic function and radial velocity at MVO ($V_{MVO}$) as a parameter of dyssynchronous motion induced by post-systolic thickening. Time-strain curves were also analyzed in the same points, and peak systolic radial strain was measured. Each parameter was expressed as the average of the 5 radial velocity or strain values in 3 consecutive cardiac cycles.

CONVENTIONAL ANALYSIS OF SYSTOLIC WALL MOTION. As a conventional measurement of regional wall motion, the percentage of change in wall thickening ($\%WT$) was measured from 3 points at the center of the risk area and the opposite normal area in the same 3 consecutive cardiac cycles as the VVI analysis, and the values were averaged. Percent WT was calculated as: $[(end\text{-}systolic\text{ myocardial thickness} - end\text{-}diastolic\text{ myocardial thickness})/end\text{-}diastolic\text{ myocardial thickness}] \times 100$.

REAL-TIME MCE. Real-time MCE (20 frames/s) was performed in the coherent contrast imaging mode (transmitting and receiving frequencies 1.75 and 3.5 MHz, respectively) using the same short-axis view as that used for the wall motion assessment. Optison (Amersham Health, Princeton, New Jersey) was diluted 1:10 in saline and administered intravenously at the rate of 1 ml/min. After a steady state of myocardial opacification was reached, ultrasound pulses at a mechanical index of 1.9 (i.e., burst frames) were transmitted for 1 s to destroy myocardial microbubbles. This was followed automatically by imaging with a mechanical index of 0.1. Myocardial contrast echocardiographic images were acquired from before the burst frames throughout the replenishment of microbubbles within the myocardium.

From real-time MCE images obtained during coronary occlusion, 1 clear image was selected during the replenishment of contrast to evaluate the ischemic risk area (16). The risk area was measured visually and expressed as a percentage of a nonperfused area in LV myocardium using Scion Image (Scion Corporation, Frederick, Maryland). The MCE analysis was performed by an observer who was blinded to the VVI data.

Experimental protocol and data analysis. To investigate sensitivity and specificity of VVI for the detection of acute myocardial ischemia, we examined 2 different ischemic conditions (flow-limiting stenosis and total occlusion) of the LCx region. First, mitral inflow derived from the apical view was recorded with the phonocardiogram. The transducer was then fixed with a mechanical arm and short-axis images were acquired at baseline. Next, the LCx was narrowed and short-axis images were obtained 2 min after creating the stenosis to stabilize the systemic and coronary hemodynamics (stenosis was tested in 14 of 18 dogs). The transducer was then removed from the mechanical arm, and mitral inflow with the phonocardiogram was recorded during stenosis, and the stenosis was subsequently relieved. After complete recovery of the wall motion to the baseline level, the transducer was fixed again and short-axis images were obtained 15 s after total LCx occlusion. To acquire the same images as baseline, the anatomical configuration of
papillary muscles and the right ventricle was adjusted carefully. Mitral inflow with the phonocardiogram was then recorded in a similar manner (i.e., after 15 s) in a second occlusion. Finally, real-time MCE images were obtained during LCx occlusion in the same short-axis view. Each dog was euthanized at the end of the experiment. The parameters $V_{SYS}$, $V_{MVO}$, peak systolic radial strain, and %WT in the center of the risk area and the opposite normal area during stenosis and occlusion were compared with values at baseline. In each parameter, sensitivity and specificity for detecting the stenosis and occlusion were also analyzed from values in the risk area only and the ratio between values in the normal and risk areas. In addition to the calculation of sensitivity and specificity, positive and negative predictive values were estimated.

To evaluate whether the spatial extent of post-systolic inward motion detected by VVI corresponds with that of the ischemic risk area, we also tested 15-s LAD occlusion after the LCx occlusion in 4 of 18 dogs. In these dogs, an interval of more than 30 min was provided for recovery from wall motion abnormality induced by the LCx occlusion. Real-time MCE images were also obtained during LAD occlusion. During LCx or LAD occlusion, $V_{MVO}$ were analyzed in 60 points of the endocardial border. The spatial extent of post-systolic inward motion was expressed as the proportion of points that indicated inward velocity vectors at MVO to the total 60 points and compared with the risk area derived from MCE.

**Interobserver and intraobserver correlations.** Twelve image clips were randomly selected from the baseline, stenosis, and occlusion conditions. To determine the interobserver correlation for the VVI parameters, the analysis was repeated by a second observer who was blinded to the values obtained by the first observer. To determine the intraobserver correlation, the analysis was repeated 2 weeks later by the same observer.

**Statistical analysis.** Data were expressed as mean ± SD. The comparison among the conditions of the LCx in hemodynamics and each functional parameter was performed by analysis of variance (ANOVA) and the post hoc Scheffé test. The comparison between values in the normal and risk areas in each functional parameter was performed by the paired t test. The receiver operating characteristic (ROC) curve was constructed for each functional parameter in differentiating the ischemic state from the nonischemic state in the risk area. The correlation between the spatial extents of post-systolic inward motion detected by VVI and the risk area derived from MCE was determined using the least-squares fit regression and Bland-Altman analyses. The interobserver and intraobserver correlations were performed by the Bland-Altman analysis. Values of p < 0.05 were considered statistically significant.

**RESULTS**

**Coronary flow and hemodynamics.** The LCx flow was 15.6 ± 6.4 ml/min at baseline and 8.6 ± 3.6 ml/min (p < 0.001 vs. baseline, a reduction of 44.3 ± 9.0%) during stenosis; flow was completely interrupted during occlusion (p < 0.001 vs. baseline and stenosis). Heart rate and systolic/diastolic blood pressure during stenosis and occlusion of the LCx were not significantly different from those at baseline (Table 1).

**Myocardial wall thickness.** In our experiment, the configuration of the LV cavity was not significantly distorted even during stenosis or occlusion, because of the short duration of myocardial ischemia. The end-systolic wall thickness in the risk area during occlusion was thinner than that at baseline. During stenosis, however, end-systolic wall thickness appeared to be relatively preserved in most dogs (Fig. 1).

**VVI and radial myocardial velocity.** Velocity vector images at mid-systole and MVO in the same dog as that shown in Figure 1 are shown in Figure 2.

### Table 1. Coronary Flow and Hemodynamics

<table>
<thead>
<tr>
<th></th>
<th>Baseline (1) (n = 18)</th>
<th>Stenosis (2) (n = 14)</th>
<th>Occlusion (3) (n = 18)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(1) vs. (2)</td>
</tr>
<tr>
<td>LCx flow (ml/min)</td>
<td>15.6 ± 6.4</td>
<td>8.6 ± 3.6</td>
<td>0 ± 0</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Heart rate</td>
<td>122 ± 21</td>
<td>124 ± 23</td>
<td>121 ± 20</td>
<td>0.982</td>
</tr>
<tr>
<td>Systolic BP (mm Hg)</td>
<td>115 ± 15</td>
<td>115 ± 14</td>
<td>111 ± 15</td>
<td>0.996</td>
</tr>
<tr>
<td>Diastolic BP (mm Hg)</td>
<td>93 ± 13</td>
<td>92 ± 12</td>
<td>88 ± 14</td>
<td>0.987</td>
</tr>
</tbody>
</table>

*Values in bold indicate statistical significance.
BP = blood pressure; LCx = left circumflex artery.*
baseline, myocardial velocity vectors indicated inward wall motion and the amplitudes of the velocity vectors were almost the same in the whole of the myocardium at mid-systole; the velocity vectors pointed entirely outward at the time of MVO. During occlusion, the inward velocity vectors at mid-systole in the risk area became smaller compared with those at baseline. Although the velocity vectors at the time of MVO were directed outward in the normal perfused area, inward velocity vectors were revealed in the risk area. During stenosis, the velocity vectors at mid-systole did not differ from those at baseline; however, outward velocity vectors at MVO in the risk area were clearly reduced compared with those at baseline.

The previously mentioned characteristics were also shown by the profiles of radial myocardial velocity at points in the risk area and the opposite
normal area (Fig. 3, which is obtained from the same dog as shown in Figs. 1 and 2). For radial velocity, positive values represent inward wall motion and negative values represent outward wall motion. At baseline, radial velocity profiles of the risk and normal areas were almost parallel, and $V_{SYS}$ and $V_{MVO}$ were similar in the 2 areas. During occlusion, $V_{SYS}$ decreased in the risk area compared with that at baseline, and inward wall motion was again revealed after AVC and reached a peak at MVO. As a result, radial velocity at mitral valve opening ($V_{MVO}$) gradually increased in proportion to the severity of ischemia. AVC = aortic valve closure; MVO = mitral valve opening.

Parameters derived from VVI and %WT. In 1 of the 18 dogs, radial strain could not be analyzed because of insufficient image quality of the epicardium. Therefore, the data of peak systolic radial strain were only obtained from the remaining 17 dogs (17 dogs for baseline and occlusion and 13 dogs for stenosis).

All functional parameters in the risk area tended to be lower than those in the normal area in peak systolic radial strain and $V_{SYS}$ and higher in $V_{MVO}$. When compared with baseline values, %WT, peak systolic radial strain, and $V_{SYS}$ in the center of the risk area significantly decreased and $V_{MVO}$ in the risk area significantly increased during occlusion ($p < 0.001$ in %WT, peak systolic radial strain, and $V_{MVO}$; $p = 0.012$ in $V_{SYS}$). Percent WT, peak systolic radial strain, and $V_{SYS}$ in the risk area tended to decrease during stenosis, but the difference did not reach the level of statistical significance. In contrast, $V_{MVO}$ in the risk area was significantly increased from that at baseline even in this condition ($p = 0.015$). Although %WT, peak systolic radial strain, and $V_{SYS}$ in the opposite normal area tended to increase during ischemia, the difference was not significant; however, $V_{MVO}$ in the normal area was significantly increased during stenosis and occlusion ($p = 0.008$ during stenosis; $p < 0.001$ during occlusion) (Table 2).

In the ROC curve analysis for detecting occlusion from values in the risk area, the areas under the curve for %WT, peak systolic radial strain, $V_{SYS}$, and $V_{MVO}$ were 0.935 ($p < 0.001$, 95% CI 0.800 to 0.989), 0.927 ($p < 0.001$, 95% CI 0.784 to 0.987), 0.809 ($p < 0.001$, 95% CI 0.643 to 0.920), and 0.985 ($p < 0.001$, 95% CI 0.874 to 0.992), respec-
Applying an optimal cutoff value of $-0.5$ cm/s for $V_{MVO}$, sensitivity was 100% and specificity was 92% (positive and negative predictive values were 98% and 97%) for detecting occlusion. For detecting stenosis from values in the risk area, the areas under the curve for %WT, peak systolic radial strain, $V_{SYS}$, and $V_{MVO}$ were 0.849 to 0.995, 0.965 (p = 0.057, 95% CI 0.488 to 0.861), 0.566 (p = 0.546, 95% CI 0.367 to 0.751), and 0.888 (p < 0.001, 95% CI 0.711 to 0.974), respectively (Fig. 4).

For detecting occlusion from the ratio between values in the normal and risk areas, the areas under the curve for %WT, peak systolic radial strain, $V_{SYS}$, and $V_{MVO}$ were 0.849 to 0.995, 0.965 (p = 0.057, 95% CI 0.488 to 0.861), 0.566 (p = 0.546, 95% CI 0.367 to 0.751), and 0.888 (p < 0.001, 95% CI 0.711 to 0.974), respectively (Fig. 4).

**Comparison of VVI and MCE.** Real-time MCE and VVI at MVO during the LCx occlusion were demonstrated in Figure 5. Myocardial contrast defect was clearly shown in the LCx region. The area indicating inward velocity vectors at MVO corresponded closely with the contrast defect area. The spatial extent of post-systolic inward motion detected by VVI significantly correlated with that of the risk area derived from MCE (linear regression: $r = 0.74$, p < 0.001, nonlinear logarithmic regression: $r = 0.79$, p < 0.001) (Fig. 6).

**Interobserver and intraobserver correlations.** For the interobserver and intraobserver correlations in the $V_{MVO}$ measurement, mean differences were $-0.03$ and $-0.07$ cm/s, and limits of agreement (+1.96 SD) were $±0.92$ and $±0.56$ cm/s, respectively. Mean differences in the $V_{SYS}$ measurement were 0.19 and 0.10 cm/s, and limits of agreement were $±0.98$ and $±1.30$ cm/s, respectively.

**DISCUSSION**

We examined the diagnostic value of parameters derived from VVI for detecting regional wall motion abnormality during flow-limiting stenosis, in which wall thickening was relatively preserved, and during total occlusion, in which contraction deteriorated. Although $V_{SYS}$ significantly decreased during occlusion, it was difficult to detect stenosis using this parameter. In contrast, $V_{MVO}$ indicated sufficient sensitivity and specificity even during stenosis. Additionally, the area indicating inward velocity vectors at MVO corresponded well with the risk area during occlusion.

**Systolic contraction during ischemia.** In the present study, an apparent deterioration in systolic wall thickening was found in the risk area during occlusion. Percentage WT and peak systolic radial strain significantly decreased; however, dyskinetic wall motion was not seen because the duration of ischemia was very short. In contrast, %WT did not significantly deteriorate during stenosis. Although...
peak systolic radial strain was significantly lower in the risk area than that in the normal area during stenosis, the deterioration in the risk area was not significant when compared to baseline because lower strain values had already been shown even at baseline. This result implies that systolic wall thickening was relatively preserved during stenosis in this study.

The relationship between the decreases in myocardial blood flow (MBF) and regional segment length shortening is fit by an exponential expression. In an experimental study by Vatner (17), the relationship between percent change in MBF and segment length was best described as segment length (\(\% \Delta\)) = \(-161.6e^{-0.047MBF(\% \Delta)}\). At 50% of control MBF, the percent decrease of segment length is about 15% in this equation. In our data, the percent change in shortening was about 12% in %WT and 17% in peak systolic radial strain during stenosis in which the decrease in coronary blood flow was 44%. This result seems to be consistent with Vatner’s equation (17). Previous studies (14,18) have also shown that systolic myocardial strain is scarcely impaired at this level of stenosis. These results suggest that the evaluation of systolic wall contraction is not sensitive for detecting hypoperfusion of the myocardium induced by flow-limiting stenosis.

Myocardial velocity by VVI during ischemia. The parameters of regional myocardial function obtained using the tissue Doppler technique cannot be analyzed in several regions of the myocardium because of angle dependency (5–7). This limitation is fatal for the diagnosis of ischemic heart disease because the impairment of wall motion occurs regionally. The newly developed technique of VVI, which uses a tissue tracking algorithm, displays velocity vectors in all segments of the myocardium without angle dependency (11–13). The vector expression of myocardial velocity permits the easy detection of dysynchronous motion of the myocardium. These characteristics of VVI may be advantageous in detecting regional wall motion abnormality due to ischemic heart disease.

In our results, \(V_{SYS}\) derived by VVI significantly decreased in the risk area during occlusion; however, like %WT and peak systolic radial strain, it did not change during stenosis when compared to baseline. Several studies have reported that tissue Doppler velocity during systole cannot distinguish the ischemic area from normal myocardium (19–22). The failure of \(V_{SYS}\) to detect stenosis in our tissue tracking data is consistent with these previous studies. In contrast, \(V_{MVO}\) increased in the risk area not only during occlusion, but also during stenosis. The increase in \(V_{MVO}\) is considered to be caused mainly by post-systolic thickening in ischemic myocardium. The ROC curve analysis demonstrated that \(V_{MVO}\) could detect myocardial ischemia with better sensitivity and specificity than %WT, peak systolic radial strain, and \(V_{SYS}\).

**Figure 4. Receiver-Operating Characteristic Curve Analysis**

Receiver operating characteristic curves of the percent change in wall thickening (%WT), peak systolic radial strain, \(V_{SYS}\), and \(V_{MVO}\) for the detection of occlusion and stenosis from values in the risk area only and the ratio between values in the normal and risk areas. The \(V_{MVO}\) parameter could detect myocardial ischemia with better sensitivity and specificity than %WT, peak systolic radial strain, and \(V_{SYS}\). Abbreviations as in Figure 3.

**Figure 5. Comparison of MCE and VVI**

Images at MVO derived from myocardial contrast echocardiography (MCE) and VVI during occlusion of the left circumflex artery in a representative dog. The area indicating inward velocity vectors at MVO corresponded closely with the contrast defect area derived from MCE. Abbreviations as in Figure 2.
VVI seems to be reasonable for the diagnosis of ischemic heart disease.

Comparison of the normal and ischemic regions. Recently, Claus et al. (23) have reported that post-systolic thickening is the result of the difference in contractility with the adjacent normal regions. Our results seem to support their theory because there was the significant difference in values of peak systolic radial strain between the normal and risk areas during ischemia. In the normal area, the increasing tendency of %WT, peak systolic radial strain, and VSYS is thought to be due to a compensatory motion. Because peak systolic radial strain and VSYS in the risk area significantly decreased during stenosis when compared to the normal area, the comparison of the normal and risk areas seemed to be still effective for detecting ischemia using these parameters. However, sensitivity and specificity of the ratio between values in the normal and risk areas was not enough for detecting stenosis in these parameters.

VMVO significantly decreased in the normal area during stenosis and occlusion. We speculate that one of the reasons is hyperkinetic dilatation in the normal area and another is prolonged isovolumetric relaxation time during ischemia. The ratio of VMVO between the normal and risk areas also demonstrated better sensitivity and specificity for detecting ischemia such as the evaluation in the only risk area. The visual assessment of velocity vectors at MVO also appeared to be useful for the easy detection of dyssynchronous motion as shown in Figure 2.

Spatial extent of post-systolic inward motion by VVI during ischemia. In patients with ischemic heart disease, the assessment of spatial extent of ischemic myocardium is important for diagnosing the jeopardized vessel and evaluating the risk stratification. Although VVI can analyze regional wall motion in all segments of the myocardium without angle dependency, it has been unclear whether the assessment of the spatial extent of ischemic myocardium is possible using this technique. In the present study, the spatial extent of the area indicating inward velocity vectors at MVO correlated well with that of the risk area derived from MCE with a linear regression. This result suggests that the analysis of post-systolic inward motion by VVI is valuable not only for sensitive detection of myocardial ischemia, but also for estimating the spatial extent of the risk area. The discrepancy between perfusion and wall motion abnormalities may be explained by the functional border zone, which is the area of nonischemic but asynergic myocardium (24, 25).

Study limitations and clinical implications. Myocardial velocity is always influenced by overall heart motion and tethering effects. The myocardial velocity derived by VVI is also influenced by these effects. Although it is suggested that post-systolic inward motion by VVI is mainly due to post-systolic thickening, it can also be induced by the overall motion of the heart. Strain rate and strain analyses of the myocardium are superior because these effects are negligible for such deformation parameters (19–22). However, we think that the
evaluation of myocardial velocity vectors at MVO is useful for detecting myocardial ischemia if the overall motion of the heart is not significant. Although Skulstad et al. (22) reported that tissue Doppler strain analysis was superior to tissue Doppler velocity for quantifying regional myocardial function, their data indicated that measurement of tissue Doppler velocity during the isovolumic relaxation time could detect myocardial ischemia. Furthermore, the assessment of myocardial velocity by VVI has some advantages; for example, tracking of the epicardium by tissue tracking is sometimes difficult in clinical settings because the tracking algorithm suffers from the surrounding noise. Strain analysis by tissue tracking may not work well in this case; nevertheless, VVI data can be analyzed.

It is not clear as to when the post-systolic inward motion should be assessed: at the time of MVO or during isovolumic relaxation. The peak velocity of the inward motion occurred not at MVO, but during isovolumic relaxation in cases of flow-limiting stenosis, as shown in Figure 3. Evaluation during isovolumic relaxation may be preferable for detecting small post-systolic motion. However, post-systolic thickening is sometimes observed even in healthy subjects (26,27). Assessment during the isovolumic relaxation may make differentiation between normal and ischemic post-systolic motion difficult. Thus, we used MVO as the time to detect dysynchronous motion caused by post-systolic thickening because ischemic post-systolic thickening occurs later than that observed in healthy subjects (26,27). Consequently, VMMVO was effective in detecting myocardial ischemia induced by stenosis.

CONCLUSIONS

The assessment of myocardial velocity vectors at the time of MVO permits easy detection of dysynchronous wall motion during myocardial ischemia that cannot be diagnosed by the conventional measurement of systolic wall thickening. Moreover, the spatial extent of inward wall motion at MVO indicates the size of the risk area. VVI may enhance the accuracy of echocardiography for diagnosing ischemic heart disease.

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REFERENCES


APPENDIX

For an online figure describing the determination of the timing of aortic valve closure and mitral valve opening, please see the online version of this article.