**Objective:** The aim of this study was to prospectively evaluate the effect of percutaneous coronary intervention in the acute period on left ventricular dyssynchrony in ST-segment elevation myocardial infarction patients by using Tissue Synchronization Imaging.

**Methods:** Forty-four ST-segment elevation myocardial infarction (MI) patients (29 male, 15 female), who were admitted within the first 12 hours of chest pain symptoms, were enrolled in the study. According to the localization of MI, the patients were divided into groups as anterior MI (n=26) and inferior MI (n=18). All echocardiography measurements were taken just before percutaneous coronary intervention (PCI) and following PCI at a mean of 3-6 days. They were assessed according to the time to reach the peak systolic velocity, which was calculated by the tissue synchronization imaging method from four pairs of non-apical alternate segments. The difference between the duration to reach the peak systolic velocity in alternate segments was regarded as left ventricle dyssynchrony and the results were compared.

**Results:** In the anterior MI group, basal anterior (p<0.01), mid-anterior segment (p<0.01) and basal septal segment (p<0.01); in the inferior MI group, the basal septal segment (p=0.02), mid-septal segment (p=0.02), and basal and mid-inferior segment (p<0.01) values were significantly lower in the post-PCI measurements when compared to the measurements taken prior to PCI. In both groups, the intraventricular dyssynchrony indices of the basal anterior-basal inferior (p<0.01), mid-anterior-mid-inferior (p<0.01) segments were found to be significantly lower in the post-PCI period when compared to the pre-PCI period.

**Conclusion:** It was found that percutaneous coronary intervention in patients with ST-elevation significantly decreases the degree of LV dysynchrony in the acute period. (Anadolu Kardiyol Derg 2014; 14: 591-8)

**Key words:** percutaneous coronary intervention, dyssynchrony, tissue synchronization imaging, ST-segment elevation myocardial infarction
system, stimulation-contraction, myocardial structure, and heart function (12). A similar relation between heart failure and myocardial synchronization has been shown in cardiac diseases such as acute myocardial infarction (2-4, 13), unstable angina pectoris (14, 15) and hypertrophic cardiomyopathy (16). The studies that demonstrate the effect of percutaneous coronary intervention on myocardial synchrony in acute myocardial infarction are limited in number. The aim of the current study was to show the effect of percutaneous coronary intervention (PCI) in the acute period on left ventricular dyssynchrony in ST-segment elevation myocardial infarction patients using Tissue Synchronization Imaging (TSI).

Methods

Study population
This study was performed in our clinics between April 2008 and June 2010. Forty-four patients (29 male, 15 female), who were diagnosed with acute ST-segment elevation acute myocardial infarction (AMI) according to ESC/ACC criteria (17), presented to our clinic within the first twelve hours of the onset of AMI, and who had an indication for PCI were included in this study. Before the measurements were taken according to the infarction area, the patients were divided into two groups as anterior AMI and inferior AMI.

Exclusion criteria:
Exclusion criteria: Previously diagnosed with heart failure, QRS duration exceeding 120 msc, cardiogenic shock, serious valvular heart disease, infarction history, uncontrolled hypertension, hypertrophic obstructive cardiomyopathy.

Patients with multiple vascular disease at coronary angiography;
Atrial fibrilasyon, prosthetic valve, pace rhythm, who had tachycardia during measurements, poor image quality, systemic diseases, chronic kidney disease.

The study was conducted in accordance with the principles of the Declaration of Helsinki and was approved by the local Ethic Committee. All patients and the healthy controls were informed about the study and their written consent forms were obtained.

Echocardiography
The first echocardiographic recordings of all the patients were obtained just before PCI in the catheterization laboratory. Standard transthoracic echocardiography measurements of all patients were taken by using a 2.5 Mhz probe of a Vivid 7 device (GE Healthcare). All images were recorded as three strokes on average for off-line measurements using the protocol of the Echopack program. Left ventricular ejection fractions were measured by a modified Simpson’s technique, and left ventricular diastolic diameter, systolic diameter, interventricular septum thickness, and posterior wall thickness were measured by using M-mode.

Tissue synchronization imaging
In the tissue Doppler imaging mode, the cursor was placed in the basal and middle regions of the lateral, septal, anterior, and inferior walls. Myocardial velocity graphics were obtained from apical-four cavity images and apical two cavity images. The apical segments of the heart were not taken into consideration in TSI since they were not coded in color in the early phases the starting point was set at the point of the opening of the aortic valves, and the end point was set at 200 milliseconds after the closure of the aortic valves, the beginning of the rapid filling period. The duration from the start of QRS to peak systolic velocity (Tscore) was measured in 8 segments (Fig. 1). The interobserver and intraobserver variabilities have been compared in 60 consecutive measurements and were 4.7% and 3.2%, respectively. Bazett’s formula was applied to remove the effect of heart rate variation on Tscore. Measurements were taken by the Bazett formula according to the heart rate with the following formula (12):

\[ T_s = \frac{\text{Tscore}}{\sqrt{R-R}} \]  
(Bazett formula)

The absolute value of the Tscore differences between alternate segments (inferior-anterior and lateral-septal) was regard-
ed as intraventricular dyssynchrony (18). Intraventricular dyssynchrony before and after PCI were calculated separately. Changes in dyssynchronization values prior to and following PCI (mean of 3-6 days) were compared in each patient to identify the differences.

Statistical analysis
All parameters were expressed as mean± standard deviation. All data were analyzed by using SPSS for Windows version 15.0 software (Chicago, IL, USA). Categorical variables were presented as frequencies and percentages continuous variables were expressed as means and SD. The normal distribution of continuous variables was tested with Kolmogorov-Smirnov test. Wilcoxon test was used to find the statistical differences in the patient groups. P values <0.05 were considered statistically significant.

Results

Clinical characteristics
The clinical and laboratory features of the study population are summarized in Table 1. Twenty-nine of the 44 patients that were enrolled in the study were male (65.9%). The average age of the patients was 58.4±9.9 years. Twenty-six of the patients (59.1%) had a diagnosis of anterior AMI and 18 were diagnosed with inferior AMI. Twelve of the patients (27.3%) had diabetes mellitus (DM), 30 (68.2%) had hypertension (HT), 25 had (56.8%) cigarette smoking history, 9 (20.5%) had hyperlipidemia, and 7 (15.9%) had a family history. Patients were admitted to our clinics within an average of 5.1±1.1 hours after the onset of chest pain. Percutaneous coronary intervention was performed on left anterior descending (LAD) artery lesions in 26 patients (59.1%), right coronary artery (RCA) lesions in 15 patients (34.1%), and left circumflex (CX) lesions in 3 patients (6.8%). Medical treatment that the patients had taken throughout the duration of their hospital stay is shown in Table 1.

Effects of PCI on the conventional echocardiographic parameters
Standard transthoracic echocardiography data of the patients were recorded at the time of referral (pre-PCI) and right before discharge (mean of 3-6 days). Left ventricular systolic diameter (LVSD) and left ventricular diastolic diameter (LVDD), left atrium dimensions, left ventricle ejection fractions (Modified Simpson), interventricular septum thickness (IVS), and posterior wall thickness (PW) were all evaluated. Pre- and post-PCI measurements were found as follows: LVSD [(34.3, 32.8) p<0.01] and LVDD [(48.5, 47.1) p<0.01], respectively. It was documented that post-PCI measurements were significantly lower compared to pre-PCI measurements. The ejection fraction was found to be significantly higher in post-PCI measurements [(37.7%, 42.9) p<0.01]. Other pre and post-PCI measurements were as follows: left atrium dimension [(38.3, 39.3) p=0.6], interventricular septum thickness [(11.7, 11.8) p=0.5] and posterior wall thickness [(11.7, 11.7) p=0.9]. There were no significant differences between the pre- and post-PCI values of these parameters. Standard transthoracic echocardiography data of the patients are shown in Table 2.

Effects of PCI on the Ts and dyssynchrony indexes
Pre- and post-PCI Ts values were measured in 8 segments. Corrections were made into two groups as the anterior AMI group and inferior AMI group according to the infarction area. In the
The current study assessed the effect of percutaneous coronary intervention on acute left ventricular dyssynchrony in ST-segment elevation myocardial infarction. Although the measurements in this study were obtained a short while after percutaneous coronary intervention, there was a significant decrease in LV dyssynchrony after PCI. Although there are many studies in the literature that investigate the effect of PCI on ST-segment elevation myocardial infarction, there aren’t studies that assess its effect on left ventricular dyssynchronization.

Echocardiography with tissue Doppler imaging (TDI) is the most commonly used method for the detection of LV dyssynchrony. Arita et al. (19) reported a significant increase in LV dyssynchrony, which was demonstrated by calculating the standard deviation of the time to peak radial strain at six mid-ventricular segments in canine models with heart failure. Karakaş et al. (20) have demonstrated the relation between non-dipper blood pressure and LV dyssynchrony in both normotensive and hypertensive individuals by using tissue Doppler imaging method. In

### Table 2. Standard transthoracic echocardiographic measurements of the patients

<table>
<thead>
<tr>
<th></th>
<th>Pre-PCI (n=44)</th>
<th>Post-PCI (n=44)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>LVDD, mm</td>
<td>48.5</td>
<td>47.1</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>LVSD, mm</td>
<td>34.3</td>
<td>32.8</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>LVEF (%)</td>
<td>37.7</td>
<td>42.9</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>PW, mm</td>
<td>11.7</td>
<td>11.7</td>
<td>NS</td>
</tr>
<tr>
<td>IVS, mm</td>
<td>11.8</td>
<td>11.7</td>
<td>NS</td>
</tr>
<tr>
<td>LA, mm</td>
<td>38.3</td>
<td>39.3</td>
<td>NS</td>
</tr>
</tbody>
</table>

IVS - interventricular septum thickness; LA - left atrium diameter; LVDD - left ventricular diastolic diameter; LVEF - left ventricular ejection fraction (Modified Simpson method); LVSD - left ventricular systolic diameter; NS - not significant; PW - posterior wall thickness

### Table 3. Average Ts values and standard deviations of segments in anterior AMI patients

<table>
<thead>
<tr>
<th></th>
<th>Pre-PCI (n=26)</th>
<th>Post-PCI (n=26)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Basal-anterior, ms</td>
<td>106.6±15.5</td>
<td>105.8±16.1</td>
<td>0.35</td>
</tr>
<tr>
<td>Basal-septal, ms</td>
<td>129.4±36.6</td>
<td>114.1±22.4</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Mid-lateral, ms</td>
<td>104.9±14.9</td>
<td>104.5±15.1</td>
<td>0.29</td>
</tr>
<tr>
<td>Mid-septal, ms</td>
<td>117.5±19.2</td>
<td>113.8±18.8</td>
<td>0.08</td>
</tr>
<tr>
<td>Basal-anterior, ms</td>
<td>174.1±18.8</td>
<td>124.7±17.6</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Basal-inferior, ms</td>
<td>106.3±17.8</td>
<td>105.2±17.1</td>
<td>0.18</td>
</tr>
<tr>
<td>Mid-anterior, ms</td>
<td>172.1±19.2</td>
<td>122.3±16.9</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Mid-inferior, ms</td>
<td>104.5±14.9</td>
<td>104.1±15.0</td>
<td>0.65</td>
</tr>
</tbody>
</table>

Ms - milliseconds; PCI - percutaneous coronary intervention

### Table 4. Pre- and post- PCI intraventricular dyssynchrony values in anterior AMI patients

<table>
<thead>
<tr>
<th></th>
<th>Pre-PCI (n=26)</th>
<th>Post-PCI (n=26)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>MAMI difference, ms</td>
<td>67.5±16.1</td>
<td>19.1±13.3</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>BABI difference, ms</td>
<td>67.7±15.2</td>
<td>19.5±12.5</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>BLBS difference, ms</td>
<td>19.1±14.7</td>
<td>19.2±13.7</td>
<td>0.78</td>
</tr>
<tr>
<td>MLMS difference, ms</td>
<td>22.1±14.0</td>
<td>22.1±13.9</td>
<td>0.92</td>
</tr>
</tbody>
</table>

MAMI - mid-anterior - mid-inferior; BABI - basal anterior - basal inferior; BLBS - basal lateral - basal septal; MLMS - mid-lateral - mid-septal; Ms - milliseconds

### Table 5. Average Ts values and standard deviations of segments in inferior AMI patients

<table>
<thead>
<tr>
<th></th>
<th>Pre-PCI (n=18)</th>
<th>Post-PCI (n=18)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Basal-lateral, ms</td>
<td>106.5±26.8</td>
<td>102.6±24.1</td>
<td>0.22</td>
</tr>
<tr>
<td>Basal-septal, ms</td>
<td>120.2±34.1</td>
<td>109.7±28.5</td>
<td>0.03</td>
</tr>
<tr>
<td>Mid-lateral, ms</td>
<td>105.9±27.4</td>
<td>101.7±21.2</td>
<td>0.22</td>
</tr>
<tr>
<td>Mid-septal, ms</td>
<td>113.2±29.7</td>
<td>108.1±29.4</td>
<td>0.04</td>
</tr>
<tr>
<td>Basal-anterior, ms</td>
<td>112.8±24.9</td>
<td>112.1±24.1</td>
<td>0.84</td>
</tr>
<tr>
<td>Basal-inferior, ms</td>
<td>172.6±43.1</td>
<td>124.4±33.8</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Mid-anterior, ms</td>
<td>111.2±25.6</td>
<td>110.1±25.5</td>
<td>0.24</td>
</tr>
<tr>
<td>Mid-inferior, ms</td>
<td>169.6±42.8</td>
<td>122.5±33.7</td>
<td>&lt;0.01</td>
</tr>
</tbody>
</table>

Ms - milliseconds; PCI - percutaneous coronary intervention

### Table 6. Pre- and post- PCI intraventricular dyssynchrony values in inferior AMI patients

<table>
<thead>
<tr>
<th></th>
<th>Pre-PCI (n=18)</th>
<th>Post-PCI (n=18)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>MIMA difference, ms</td>
<td>61.4±28.5</td>
<td>16.6±20.0</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>BIBA difference, ms</td>
<td>60.1±31.3</td>
<td>18.1±20.3</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>BLBS difference, ms</td>
<td>24.5±21.8</td>
<td>22.6±20.4</td>
<td>0.07</td>
</tr>
<tr>
<td>MLMS difference, ms</td>
<td>13.7±10.1</td>
<td>12.5±10.1</td>
<td>0.07</td>
</tr>
</tbody>
</table>

BIBA - basal inferior - basal anterior; BLBS - basal lateral - basal septal; MIMA - mid-inferior - mid-anterior; MLMS - mid-lateral - mid-septal; Ms - milliseconds
another study done by Karakaş et al. (21), by using the same method they have demonstrated that left ventricular systolic dyssynchrony is an early manifestation of heart involvement in SCA patients with normal EF and narrow QRS. Schiller et al. (22) used the TSI method to evaluate the myocardial dyssynchrony before and after the cardiac pacemaker treatment. They reported that the TSI method is an easy and applicable method in the quantitative detection of regional dyssynchrony. Penicka et al. (23) used the comparison of left ventricle segments’ duration to reach peak systolic velocity from the start of QRS to evaluate intraventricular synchronization disorder. It was reported that comparing the durations needed for two alternate walls from four apical cavities to reach peak systolic velocity (septum lateral wall delay) was satisfactory. Thus, it was claimed that a minimum delay of 60 milliseconds is sufficient for the diagnosis of distinct intraventricular mechanical synchronization disorder. Yu et al. (18) evaluated more segments together and expressed the temporal heterogeneity of segmental movement as the standard deviation of 12 basal and middle segments. They determined that having a standard deviation exceeding 33 milliseconds is an important indicator for dyssynchrony. This index, which is called the Yu index, shows a high correlation with the delay between the septum and posterior wall and parasternal short axis. In the current study, the comparison of durations that are passed till the two alternate walls reach peak systolic velocity were performed in four and two apical cavity images (septum-lateral wall delay, anterior-inferior wall delay); peak systolic velocity differences between the walls were used as intraventricular dyssynchrony indexes. In both inferior AMI patients and anterior AMI patients, before percutaneous coronary intervention, in the comparison of anterior and inferior walls, the average intraventricular dyssynchrony index was found to be higher than 60 milliseconds. Following percutaneous coronary intervention, this parameter has an approximate average of 20 milliseconds. As a result, the most distinct finding of this study, viewed in light of the present literature, is that there is significant LV dyssynchrony before percutaneous coronary intervention of acute AMI and that the dyssynchrony degree decreases significantly following percutaneous coronary intervention.

Previous studies suggested the presence and clinical relevance of LV dyssynchrony in the setting of chronic heart failure. In this group of patients, the loss of LV synchronous contraction was related to impaired LV systolic function and poor hemodynamic status, which is an indicator of a poor outcome (11, 24). Cho et al. (25, 26) demonstrated that mechanical dyssynchrony was a powerful predictor of mortality or cardiac events in heart failure patients with normal and wide QRS. Penicka et al. (11), Fauchier et al. (27), and Bader et al. (10) reported that LV dyssynchrony was prognostic of cardiac endpoints. Kutyifa et al. (28) investigated VT/VF events and LV dyssynchrony in mild heart failure patients with LBBB and an implanted CRT device and compared them to patients with non LBBB and demonstrated that improved synchrony might translate into a reduction of ventricular arrhythmic events in LBBB patients. Ludwig et al. (29) reported that patients with heart failure and LBBB, acute RVA pacing induces greater mechanical dyssynchrony and further impair LV function. Sahebjam et al. (30) investigated the relationship between left atrial function and left ventricular dyssynchrony in heart failure patients. They revealed that left ventricular dyssynchrony was independently correlated with the deformity indices of the LA lateral wall.

The assessment of LV dyssynchrony during AMI has been performed using various echocardiographic techniques such as pulse wave TDI (31), color-coded TDI (2), speckle tracking (3), real time 3D echocardiography (32), and magnetic resonance imaging (MRI) (13). Although pulsed wave tissue Doppler is faster, the spatial resolution of this method is low and it has the disadvantage of comparing the measurements which are obtained from numerous different cycles. So heart rate variation is much more sensitive to the global motion and loading status of heart. Nevertheless it is reported that similar results with colored Doppler could be obtained (1). With the measurement of tissue displacement, in other words tissue tracking imaging, the visual and quantitative amount of tissue motion can be interpreted in millimeters-centimeters in a relatively easier way (33-36). As strain and strain rate imaging are not affected by impulse-traction and translation, they may provide better differentiation of mechanical delay (37, 38). The frequently observed shortening following systole with these two methods causes the term “peaking time” to become more complicated. MRI is considered the gold standard for assessing cardiac shape and function, and other modalities such as real-time three-dimensional echocardiography (RT-3DE) (39, 40), closely correlate with MRI. Dyssynchrony using tissue Doppler images (TDI) during the acute phase of myocardial infarction is also considered a predictor of LV remodeling (3, 41) and although RT-3DE can be used to assess cardiac dyssynchrony (42), few reports have been published and the results do not coincide with those of TDI (43). The current study used TSI to detect dyssynchronous wall motion because of its ease of use and relatively good reproducibility. Starting point was set at the point of the opening of the aortic valves, and the end point was set at 200 milliseconds after the closure of the aortic valves, the beginning of the rapid filling period. In this way, the dyssynchronous motion within the systolic period was observed, whereas the latent influence of contractile motion during the isovolumic contraction period was excluded.

The patients in the current study had an initial EF of 37.7%. All had significant left ventricular systolic dysfunctions. In accordance with the outcomes of the studies in the literature, the patients of this study had distinct left ventricular dyssynchrony and their EF increased to 42.9% following percutaneous coronary intervention, while there was a significant decrease in left ventricular dyssynchrony. Different patterns were observed in pre PCI and post-PCI T1s values in anterior AMI and inferior AMI groups according to the infarction area. As expected in anterior AMI, the T1s values of the anterior segments in the post-PCI decreased. On the other hand, inferior segments were
affected by inferior infarction and the Ts values decreased in the post-PCI. The decrease of the Ts values in the basal septal segments is somewhat intriguing. This could imply that the septal wall of the LV is more vulnerable to develop dysynchrony in the occurrence of LV systolic dysfunction. Manka et al. (44) demonstrated that the regression of left ventricular dys synchrony during healing of acute AMI. Zhang et al. (2) analyzed left ventricular systolic dys synchrony in acute myocardial infarction patients with normal QRS durations. A total of 47 ST-segment elevation myocardial infarction patients were enrolled in the study and compared to the control group. Peak systolic velocity durations were found to be significantly longer in the AMI group. Again in this study, peak systolic velocity durations of anterior AMI patients was compared to those of inferior AMI patients and was found to be longer. A correlation between the degree of LV dys synchrony and infarct area was detected. Nucifora et al. (45) investigated the effect of post-MI left ventricular functions on dys synchrony in their study. Left ventricular dys synchrony was found to be higher in patients with damaged left ventricle functions, large infarct areas, and in anterior AMI. However in these studies, there were no pre- or post-PCI comparisons made.

Finally, the accuracy and superiority of the TSI technique in the dysynchrony of acute myocardial ischemia was demonstrated in a fewer number of clinical studies (46-49). The current study is important because myocardial dys synchrony can be determined quantitatively in patients with AMI in the acute ischemic period and in the post-PCI period. The current results demonstrate the potential clinical advantages of using TSI in the detection of myocardial dys synchrony changes in AMI patients in the early reperfusion period.

**Study limitations**

The present study has several limitations. Only longitudinal myocardial motion and dys synchrony were examined in the present study, whereas radial and circumferential motions were not evaluated. The reproducibility and usefulness of TDI in the evaluation of mechanical dys synchrony has been questioned recently (50). Recently the new methods such as speckle tracking imaging and three dimensional echocardiography were not used. The other limitation was the image quality and the artifacts. Its generation of a difference between the first hour admission and 12th hour admission in terms of myocardial damage, may effect the left ventricle dys synchrony. The technique is highly dependent on the adequate training of operators and its reproducibility remains high in experienced laboratories. The measurements were done in the early period. The late results were not evaluated. In the near future, technique-related limitations can be overcome by the development of 2- and 3-dimensional or intra-cardiac strain echocardiography methods.

**Conclusion**

This study demonstrated that percutaneous coronary intervention greatly decreased LV dyssynchrony in the acute period in patients with acute ST-segment elevation acute myocardial infarction. The deleterious effect of systolic asynchrony on global LV function may contribute to the acute remodeling process after AMI and one of the treatment targets in these patients should be LV dys synchronization. There are many studies required in order to use this method routinely in LV dys synchronization.

**Conflict of interest:** None declared.

**Peer-review:** Externally peer-reviewed.


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