Internal Medicine and Medical Investigation Journal
E-ISSN: 2474-7750
Homepage: www.imminv.com

ORIGINAL ARTICLE

# Evaluation of Longitudinal Tissue Velocity and Deformation Imaging in Akinetic Non-viable Apical Segments of Left Ventricular Myocardium

Hakimeh Sadeghian[1], Parastoo Vosoughi[2], Afsaneh Sadeghian[3], Masoumeh Lotfi-Tokaldany[2], Mohammad Moein Ashrafi[4], Soraya Shahrzad[1]*

[1]*Shariati Hospital, Tehran University of Medical Science, Tehran, Iran*

[2]*Tehran Heart Center, Tehran, Iran*

[3]*Bahar Hospital, Shahrood University of Medical Science, Shahrood, Iran*

[4]*Islamic Azad University, Yazd, Iran*

**Corresponding Author:** Soraya Shahrzad, E-mail: sshahrzad@yahoo.com

**ARTICLE INFO**

*Article history*
Received: ???
Accepted: ???
Published: ???
Volume: x
Issue: x

Conflicts of interest: None
Funding: None

*Key words*
*Tissue Doppler Imaging,*
*Myocardium,*
*Viability,*
*Strain Rate (SR) Imaging,*
*Non-viable Apical Segments*

**ABSTRACT**

**Introduction:** The use of tissue velocity and strain rate imaging is proposed for the quantification of non-viable myocardium. This study is aimed at investigating the differences in tissue velocity and strain rate imaging indices between non-viable left ventricular apical segments and the normal segments using segment-by-segment comparison. **Materials and Method:** Thirty-two patients with akinetic left ventricular apical segments and without viability were selected using two-dimensional echocardiography and dobutamine stress echocardiography; 32 individuals with normal echocardiography and coronary angiography formed the normal group. Peak systolic velocity, peak systolic strain, and strain rate were measured in the four left ventricular apical segments and the apex 17$^{th}$ segment. **Results:** The patient group had a significantly lower ejection fraction (26.88±6.06% vs. 56.56±2.36%; p<0.001). Overall, the patient group had significantly lower resting peak systolic velocity, systolic strain, and strain rate. In the segment-by-segment comparison, only systolic strain showed a remarkable reduction in the patient group, while reduction in Sm and strain rate were not significant in all the segments. After dobutamine stress echocardiography, only systolic strain showed an insignificant increase compared to the resting values. In the apex 17$^{th}$ segment, Sm showed significant reduction in the patient group. **Conclusion:** The ST in apical segments may be used as a quantitative index for detecting akinesia both at rest and after dobutamine infusion. Reduction in Sm can be used as a marker of akinesia in the apical cap at rest.

## INTRODUCTION

Two types of Doppler-based modalities, namely tissue velocity imaging and strain rate imaging are currently in use for the quantitative assessment of the myocardial regional systolic function. Strain and strain rate imaging have been introduced to assess the myocardial shortening fraction and shortening rate, which is an advantage over tissue velocity imaging (1-3). Experimental and clinical studies have demonstrated that peak systolic velocity and strain rate have the potential to discriminate acute from chronic ischemia and myocardial infarction (4-8).

Although the individual left ventricular (LV) segments have their own specific motion and characteristics, the conclusions of these studies were focused on the overall changes of the LV myocardial walls. In our previous reports, we compared the velocity and deformation indices of inferobasal non-viable akinetic (9) and aneurismal (10) segments with the same indices in normal individuals. The tissue velocity imaging and strain rate imaging parameters decreased significantly and the range of peak systolic strain rate (SR) for the non-viable akinetic segment did not overlap with that of the normal segments. There was no overlap in the peak systolic strain (ST) and SR between aneurismal and normal inferobasal segments (10). Accordingly, the resting strain rate had the potential to discriminate between the non-viable inferobasal and normal segments in a non-invasive way. As there is a wide variation in ST, SR and Sm (peak systolic inward motion) between each of the LV walls and basal levels, segment-by-segment analysis may produce a chance for finding little differences in the figures and help to discriminate between normal conditions and the diseased state.

Based on our previous studies (9, 10), here, we aimed to compare the velocity imaging and strain rate imaging indices in non-viable akinetic apical and apex 17$^{th}$ segments with normal apical segments. The modalities that differ between akinetic non-viable apical segments and the normal

Published by Mehrabani Publishing LLC.

http://dx.doi.org/10.24200/imminv.v3i4.1

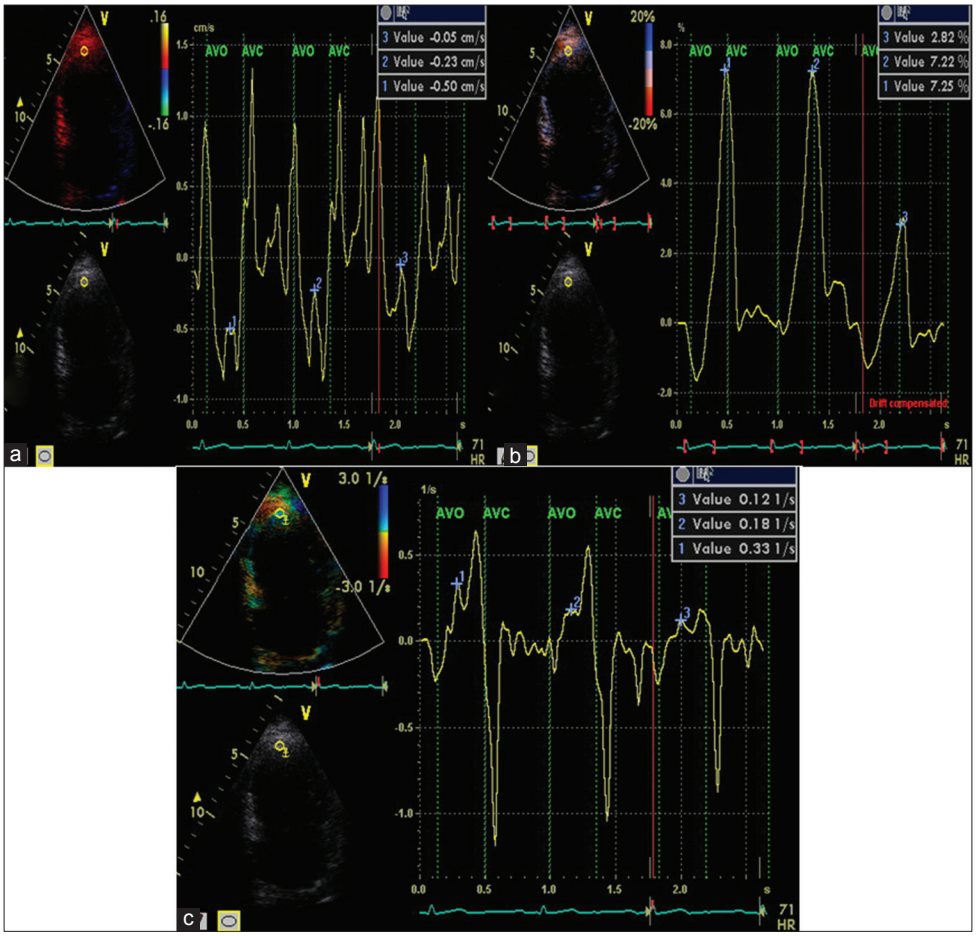


**Figure 1:** Measurement of Sm (A), Strain (B) and Strain rate (C) in apex 17 sgement.

segments were also determined by dobutamine stress echocardiography.

## MATERIALS AND METHODS

Thirty-two patients who had undergone dobutamine stress echocardiography (DSE) for the detection of myocardial viability between March 2013 and July 2015 were included in this study as the case group. The patients were selected from both in-hospital and out-patients that were referred for DSE and exhibited apical akinesia on echocardiography. These patients had akinesia in 5 LV segments (4 apical and apex 17$^{th}$) in resting two-dimensional echocardiography that did not show improvement in wall motion grading after dobutamine injection. Subsequently, 32 normal individuals with normal angiography, normal coronary arteries, and normal two-dimensional echocardiography were recruited as the control group. Baseline characteristics were obtained from the cases and controls and transcribed into a computerized format. This study was approved by our institutional committee on human research. Informed consent was obtained from all the patients before being included in the study.

Two-dimensional echocardiography, tissue velocity, and strain rate imaging were performed for the study populations with the Vivid 7 Dimension ultrasound system (GE Healthcare). The two-dimensional echocardiography was applied according to the American Society of Echocardiography (ASE) published guidelines. The details of the tissue velocity imaging and strain rate imaging carried out in our department were published in the previous reports (9, 10). Briefly, three heart cycles of the apical four, three, and two chamber views were captured by conventional two-dimensional echocardiography and color tissue Doppler mode. The frame rate was above 100 ms for tissue Doppler imaging. A 17-segmental model of the LV was used for all the analyses; off-line analysis was performed by an expert echocardiographer.

The ST, SR, and Sm were measured at rest and peak stress during dobutamine study in 4 apical and apex 17$^{th}$ segments, and the values for ST, SR, and Sm were expressed as percentage (%), seconds$^{-1}$ (s$^{-1}$), and cm/s, respectively (Figure 1).

The parameters of the LV apical segments were compared between the non-viable akinetic and normal groups. The same parameters were also compared before and after the dobutamine study. The patterns of the strain curve in systole were either deemed as shortening (descending with decreasing pattern) or expansion (ascending with increasing pattern.

The reproducibility of the tissue velocity and strain rate imaging parameters in our institute has been published previously (9).

Dobutamine stress echocardiography: Echocardiography was performed under resting condition and during each dobutamine infusion step as described previously (9, 11).

**Table 1.** Comparison between normal and case groups according to basic characteristics and two-dimensional echocardiographic characteristics

| | Patient group (n=32) | Normal group (n=32) | P value |
|---|---|---|---|
| Male sex | 28 (87.5) | 26 (81.3) | 0.491 |
| Age | 61.81±10.24 | 60.25±6.43 | 0.468 |
| NYHA functional | | | <0.001 |
| class I | 3 (9.4) | 15 (46.9) | |
| class II | 17 (53.1) | 17 (53.1) | |
| class III | 37.5 | 0 | |
| Typical chest pain | 22 (68.8) | 1 (3.1) | <0.001 |
| Family history of coronary artery disease | 4 (12.5) | 5 (15.6) | 0.799 |
| Smoking | 19 (59.4) | 14 (43.8) | 0.211 |
| Hyperlipidemia | 15 (46.9) | 13 (40.6) | 0.614 |
| Hypertension | 20 (62.5) | 10 (31.3) | 0.012 |
| Diabetes mellitus | 15 (46.9) | 5 (15.6) | 0.007 |
| EF | 26.88±6.06 | 56.56±2.36 | <0.001 |
| LVDd | 60.56±6.51 | 46.38±4.31 | <0.001 |
| LVSd | 47.06±8.29 | 29.63±5.42 | <0.001 |
| Interventricular septal diameter | 9.72±2.11 | 9.91±1.79 | 0.703 |
| Posterior wall diameter | 9.25±1.11 | 9.88±1.77 | 0.092 |

Data are expressed as percentages or mean±SD. New York Heart Association functional classification, EF: ejection fraction, LVDd: left ventricular end diastolic diameter, LVSd: Left ventricular end systolic diameter

Beta blockers, calcium antagonists, and nitrates were discontinued in the patients, at least 2 days before dobutamine study.

After baseline echocardiography, dobutamine was infused intravenously, beginning at 5 μg/kg/min and increased up to 15 μg/kg/min with three minutes interval between the doses. With the occurrence of severe hypotensive or hypertensive response, significant arrhythmia, prolonged angina, significant electrocardiographic changes, or completion of the protocol, the infusion was terminated. The echocardiographic images were analyzed off-line, using a seventeen-segment model. Segmental wall motion was scored on a four-point scale: 1- normal, 2- hypokinetic (severely reduced wall thickening and inward wall motion), 3- akinetic (absence of systolic thickening and wall motion), and 4- aneurismal (dyskinetic regions with diastolic contour abnormality). Demonstration of wall thickening in a previously akinetic segment or normalization of thickening in a previously hypokinetic segment was considered as the criteria for myocardial viability (11, 12, 13).

Statistical analysis: The continuous variables are presented as mean ± standard deviation, while the categorical variables are summarized by absolute frequencies and percentages. Continuous variables were compared among two groups using the Student's t-test or the nonparametric Mann-Whitney U test whenever the data did not appear to have normal distribution, and categorical variables were compared using the chi-square or Fisher's exact test, as required. Changes in the mean of segments before and after stress in the non-viable group were tested using paired-t test or the Wilcoxon signed-rank test as appropriate. The statistical package for the social sciences (SPSS v. 15.0, Inc., Chicago, IL) software was used for the statistical analyses. *P*-values≤0.05 were considered statistically significant.

## RESULTS

There was no significant difference in the mean age of the patient (61.81 ± 10.24 years) and the normal (60.25 ± 6.43 years) group. The patient group had a significantly lower ejection fraction compared to the normal group (26.88 ± 6.06% vs. 56.56 ± 2.36%; p<0.001). The comparison of the baseline characteristics between the two groups is shown in Table 1. The frequency of chest pain and diabetes mellitus was more in the patient group compared to the normal group.

Comparison between akinetic and normal groups at rest: Conventional visual wall-motion assessment and quantitative analysis of strain, strain rate curves, and peak systolic velocity were carried out in all the patients.

Resting Sm: Overall, the four non-viable segments in the apical level had a significantly lower mean peak Sm than the corresponding normal segments at rest (Table 2). Segment-by-segment comparison in the apical level showed a remarkable decrease in Sm in each of the individual apical non-viable segments, except the septal apical. The range of Sm in each non-viable segment overlapped with that of the normal ones.

Resting ST: The mean value of ST was significantly lower in the akinetic non-viable group, either overall or in the segment-by-segment comparison (Table 2). The range of ST in the individual non-viable segments overlapped with those of the normal ones.

Resting SR: Considering all the apical segments, the overall SR in the four apical segments had a remarkable decrease

**Table 2.** Tissue velocity and strain rate imaging parameters in non-viable apical segments compared to normal segments at rest

| | Normal (n=32) | Non-viable (n=32) | P value* |
|---|---|---|---|
| Septal apical | | | |
| ST (%) | –17.66±6.42 | –7.19±9.52 | <0.001 |
| SR (1/s) | –0.63±0.44 | –0.47±0.57 | 0.222 |
| Sm (cm/s) | 2.17±1.11 | 1.62±1.42 | 0.091 |
| Lateral apical | | | |
| ST (%) | –11.28±7.06 | –1.29±8.40 | <0.001 |
| SR (1/s) | –0.61±0.74 | –0.40±0.56 | 0.122 |
| Sm (cm/s) | 2.36±1.46 | 0.66±1.42 | <0.001 |
| Anterior apical | | | |
| ST (%) | –10.26±7.13 | –2.30±6.66 | <0.001 |
| SR (1/s) | –0.38±0.37 | –0.30±0.40 | 0.420 |
| Sm (cm/s) | 2.09±1.53 | 0.62±1.32 | <0.001 |
| Inferior apical | | | |
| ST (%) | –18.44±7.49 | –3.95±8.43 | <0.001 |
| SR (1/s) | –0.71±0.38 | –0.62±0.75 | 0.561 |
| Sm (cm/s) | 2.45±1.26 | 0.421±2.08 | <0.001 |
| Apex 17th segment | | | |
| ST (%) | –4.84±11.57 | –4.42±6.91 | 0.860 |
| SR (1/s) | –0.46±0.48 | –0.54±0.81 | 0.612 |
| Sm (cm/s) | 1.24±0.98 | 0.64±0.60 | 0.004 |
| Overall four apical segments | | | |
| ST (%) | –14.44±7.87 | –3.72±8.54 | <0.001 |
| SR (1/s) | –0.58±0.43 | –0.45±0.59 | 0.042 |
| Sm (cm/s) | 2.27±1.34 | 0.84±1.64 | <0.001 |

Data are presented as mean±SD. ST: Strain, SR: Strain rate, Sm: Peak systolic inward motion (cm/s)

from -0.58±0.43 to -0.45±0.59 (p=0.042). Although the mean SR of the individual segments reduced in the non-viable segments, this reduction was not statistically significant (Table 2). The range of SR in each non-viable segment overlapped with that of the normal ones.

Apex 17th segment: The akinetic non-viable apex 17th segment also showed a significant decrease in Sm. The values of ST and SR in the apex 17th segment were not significantly different between the non-viable and normal groups. The ranges of values for Sm, ST, and SR in the apex 17th non-viable segments overlapped with those of the normal segments. The ST and SR had negative values both in the normal and akinetic non-viable groups.

Pattern of curve: Among the non-viable segments, 31 out of 128 (24.2%) segments showed an ascending curve, and the rest showed a descending curve as follows: 10 (31.2%) of the anterior apical, 9 (28.1%) of the lateral apical, 7 (21.8%) of the inferior apical, and 5 (15.6%) of the septal apical segments had ascending curves. In the patient group, 11 (34.4%) apex 17th segments showed ascending curve and the rest had descending curve. In the normal group, ascending curve was observed in 8 (32%) apex 17th segments (p=0.412). In the other 4 apical segments, all the septal apical and inferoapical segments had a descending curve, while 2 (6.5%) anteroapical and one (3.1%) lateral apical segment showed an ascending curve; the others had a descending curve.

Comparison before and after stress in non-viable segments: The mean values of the study parameters for each segment and the overall apical segments before and after dobutamine stress test are presented in Table 3. When all four apical segments were considered together, the increase in the mean value for Sm was remarkable, whereas it was non-significant for ST and SR. In most of the non-viable segments, after dobutamine infusion, the mean values of Sm, ST, and SR had an unremarkable rise. The mean increase was only significant for SR in the septal apical and for Sm in the lateral and inferior apical segments. In the apex 17th segment, no significant changes in ST, SR, and Sm was observed. Although ST showed an increase in all 4 apical and apex 17th segment after DSE, it was insignificant.

## DISCUSSION

The results of the present study indicated that the reduction in Sm, ST, and SR was significant for the four apical non-viable segments. In our segment-by-segment comparison, each individual apical segment showed a significant decrease in ST, which after DSE, exhibited a non-significant increase; this reduction for SR was not remarkable. In the apex 17th non-viable segment, Sm decreased at rest.

Till date, little attention had been paid to the evaluation of the velocity and deformation indices in individual LV segments. Segment-by-segment comparison between normal and functionally impaired LV segments may reveal different characteristics of the segments as the geometry of the LV myocardium differs in different walls and basal levels (14).

It is noteworthy that in the inferobasal non-viable akinetic segments, the reduction in ST and SR was significant (9) and the range of SR did not overlap with that of the normal segments, whereas in the apical non-viable segments, the range of ST and SR overlapped with these values of the normal subjects. This observation indicated that the segments of the inferior wall and the basal level had different individual characteristics. The values of SR and ST for the akinetic inferobasal segments were -0.39 ± 0.20 $s^{-1}$ and -3.86 ± 4.12%, respectively (9); in the present study, the mean value of the overall apical segments was -0.45± 0.59 $s^{-1}$ for SR and -3.72 ± 8.54% for ST.

Here, we found that Sm was lower in the non-viable apical segments than in the normal apical ones, which is in agreement with the previous reports (9, 15, 16, 17). Localizing in specific LV segments, we reported a mean Sm of 3.58 ± 1.08 cm/s for the non-viable inferobasal segments (9), while this value was 0.84±1.64 cm/s for the apical segments (this study).

Regarding the usefulness of tissue velocity and strain rate imaging during viability assessment by DSE, Weidemann et al. (18) recently found significant reduction in radial peak systolic strain and strain rate in non-transmural

**Table 3.** Tissue velocity and strain rate imaging parameters in non-viable apical segments before and after stress

| | Rest | Stress | Mean difference | 50th percentile for difference | P value** |
|---|---|---|---|---|---|
| Septal apical | | | | | |
| ST (%) | –7.19±9.52 | –8.45±10.30 | –1.55±11.11 | –2.00 | 0.458 |
| SR (1/s) | –0.47±0.57 | –0.74±0.49 | 0.27±0.70 | –0.10 | 0.036 |
| Sm (cm/s) | 1.62±1.42 | 1.99±1.78 | 0.37±1.12 | 0.17 | 0.070 |
| Lateral apical | | | | | |
| ST (%) | –1.29±8.40 | 0.52±13.63 | 2.48±13.45 | 2.00 | 0.329 |
| SR (1/s) | –0.40±0.56 | –0.24±0.84 | 0.17±1.00 | 0.10 | 0.353 |
| Sm (cm/s) | 0.66±1.42 | 1.28±1.35 | 0.65±1.39 | 0.40 | 0.014 |
| Anterior apical | | | | | |
| ST (%) | –2.30±6.66 | –0.35±0.77 | 0.96±9.02 | 0 | 0.070 |
| SR (1/s) | –0.30±0.40 | –0.41±0.83 | 0.09±0.79 | –0.10 | 0.541 |
| Sm (cm/s) | 0.62±1.32 | 0.99±1.10 | 0.39±1.31 | 0.10 | 0.115 |
| Inferior apical | | | | | |
| ST (%) | –3.95±8.43 | –8.0±8.275 | –4.04±11.62 | –4.55 | 0.077 |
| SR (1/s) | –0.62±0.75 | –0.59±0.37 | 0.05±0.75 | –0.10 | 0.716 |
| Sm (cm/s) | 0.421±2.08 | 1.63±1.57 | 1.19±2.41 | 0.42 | 0.012 |
| Apex 17th segment | | | | | |
| ST (%) | –4.42±6.91 | –1.93±9.89 | 2.92±10.71 | 1.00 | 0.154 |
| SR (1/s) | –0.54±0.81 | –0.63±1.17 | –0.21±1.00 | 0 | 0.268 |
| Sm (cm/s) | 0.64±0.60 | 0.73±0.92 | 0.08±0.78 | 0 | 0.581 |
| Overall four apical segments | | | | | |
| ST (%) | –3.86±8.22 | –3.99±10.64 | 0.53±11.58 | 1 | 0.626 |
| SR (1/s) | –0.47±0.64 | –0.52±0.80 | 0.04±0.83 | 0.1 | 0.613 |
| Sm (cm/s) | 0.80±1.49 | 1.34±1.44 | 0.64±1.65 | -0.25 | <0.001 |

Data are presented as mean±SD. ST: Strain, SR: Strain rate, Sm: Peak systolic inward motion (cm/s)

infarction and no further change in these indices with dobutamine infusion in an experimental model. They concluded that the transmural extension of the scar could be defined by the regional deformation response. Similarly, a previous report (19) showed an approximate 100% increase in SR during dobutamine study in viable segments (determined by 18-FDG-PET) and no change in the non-viable segments. In line with their finding, we found that the mean increase in ST was not significant in all 4 apical segments.

According to Hoffmann et al., the mean increase in Sm in non-viable segments after stress was 1.08 ± 0.86 cm/s. Cain et al. (20) reported a relatively lower mean increase in Sm (0.3 ± 0.2 cm/s) in non-viable segments after dobutamine study. For the apical region, the value for Sm was 0.64 ± 1.65 cm/s in our study.

In our evaluation of the apex 17th segment, a reduction in Sm in the akinetic non-viable segments was observed, but there was no reduction in ST and SR. Additionally, 11 (34.4%) non-viable and 8 (25%) normal apex 17th segments had an ascending curve in resting strain rate imaging.

In a previous evaluation of inferobasal non-viable akinetic segments (10), SR showed the potential for discriminating non-viable segments from the normal segments, whereas in the present study, Sm, ST, and SR in the apical segments did not show this ability. The Sm and ST decreased significantly in the non-viable apical segments compared to the normal apical segments, but the ranges of these indices overlapped, disabling them in introducing a cut-off value for discriminating the non-viable from the normal segments. In the apex 17th segment, the reduction in Sm in the non-viable segments was remarkable, whereas the deformation indices did not show significant changes. These observations may hypothesize the difference between the basal and the apical level of the myocardium when it comes to their quantification by deformation indices. Therefore, future studies on the different segments of the mid-portion and other basal akinetic segments as well as aneurismal segments in various myocardial levels may reveal different SRI profiles.

Only a few studies regarding the pattern of the curve in the different segments are available. Inferobasal aneurismal segments had upward systolic strain, while all the inferobasal normal segments showed a descending curve (10). Among the non-viable inferobasal segments, 28% had ascending and 72% had descending curves, while all the normal segments had a descending curve (9). In the apex 17th segment, as already mentioned by professor Marwick (21), short axis thickening (upward curve) was observed in the systole by strain rate imaging. Although strain rate imaging is angle de-

pendent, it can be avoided by careful acquisition (21). One of the limitations in this study was the inclusion of patients with akinetic viable apical segments; their absence could have provided a better understanding of the potential clinical utilization of SRI for the determination of myocardial function. At the time of this study, speckle tracking was not available in our center. Future studies with newer modalities might help in differentiating non-viable myocardium in different segments.

## CONCLUSION

The ST in apical segments may be used as a quantitative index for detecting akinesia both at rest and after dobutamine infusion. Reduction in Sm can be used as a marker of akinesia in the apical cap at rest. New echocardiographic modalities like speckle Two-Dimensional (2D)-echocardiography could be further evaluated for this purpose.

## ACKNOWLEDGMENTS (FUNDING SOURCE)

There is no Founding Source.

## AUTHER CONTRIBUTIONS

Hakimeh Sadeghian: Main Idea, Parastoo Vosoughi : Collecting of data, Afsaneh Sadeghian: comments, Masoumeh Lotfi-Tokaldany :writing the paper, Mohammad Moein Ashrafi: English editing and submission, Soraya Shahrzad: Main comments.

## CONFLICT OF INTEREST

None

## ETHICAL STANDARDS

The proposal was approved by research department of Tehran Heart Center.

## REFERENCES

1. Stoylen A, Heimdal A, Bjornstad K, Torp HG, Dr Techn, Skjmrpe T. Strain Rate Imaging by Ultrasound in the Diagnosis of Regional Dysfunction of the Left Ventricle. Echocardiography 1999; 16: 321-329.
2. Smiseth OA, Stoylen A, Ihlen H. Tissue Doppler imaging for the diagnosis of coronary artery disease. Current Opinion in Cardiology: 2004; 19: 421-9.
3. Citro R, Galderisi M. Myocardial Postsystolic Motion in Ischemic and Not Ischemic Myocardium: The Clinical Value of Tissue Doppler. Echocardiography 2005; 22 (6): 525-532.
4. Bach DS, Armstrong WF, Donovan CL, Muller DWM**.** Quantitative Doppler tissue imaging for assessment of regional myocardial velocities during transient ischemia and reperfusion. Am Heart J 1996; 132: 721–5.
5. Edvardsen T, Skulstad H, Aakhus S, Urheim S, Ihlen H. Regional myocardial systolic function during acute myocardial ischemia assessed by strain Doppler echocardiography. Journal of American College of Cardiology 2001; 37(3): 726–30.
6. Derumeaux G, Ovize M, Loufoua J, Lofoua J, Andre-Fouet X, Minaire Y, et al. Doppler tissue imaging quantities regional wall motion during myocardial ischemia and reperfusion. Circulation 1998; 97: 1970–7.
7. Kukulski T, Jamal F, D'Hooge J, Bijnens B, De Scheerder I, Sutherland GR. Acute changes in systolic and diastolic events during clinical coronary angioplasty: a comparison of regional velocity, strain rate, and strain measurement. J Am Soc Echocardiography 2002;15:1–12.
8. Jamal F, Kukulski T, D'hooge J, De Scheerder I, Sutherland G. Abnormal postysystolic thickening in acutely ischemic myocardium during coronary angioplasty: a velocity, strain, and strain rate Doppler myocardial imaging study. J Am Soc Echocardiography 1999; 12:994–6.
9. Sadeghian H, Majidi S, Lotfi-Tokaldany M, Nikdoust F, Sheikhfathollahi M, Abbasi SH. Evaluation of longitudinal tissue velocity and deformation imaging in akinetic nonviable inferobasal segments of left ventricular myocardium by dobutamine stress echocardiography. Echocardiography 2009; 26:801-6.
10. Sadeghian H, Ahmadi F, Lotfi-Tokaldany M, Salehi R, Zavar R, Fathollahi MS, et al. Longitudinal tissue velocity and deformation imaging in inferobasal left ventricular aneurysm. Echocardiography 2010; 27: 803-8.
11. Sadeghian H, Lotfi Tokaldany M, Fallah N, Abbasi SH, Ahmadi SH, karimi AA, et al. Accuracy of Dobutamine Stress Echocardiography in Detecting Recovery of Contractile Reserve after Revascularization of Ischemic Myocardium. Journal of Tehran University Heart Center. 2007; 4: 223-228.
12. Sadeghian H, Majd-Ardakani J, Lotfi-Tokaldany M, Jahangiri C, Fathollahi MS. Comparison between dobutamine stress echocardiography and myocardial perfusion scan to detect viable myocardium in patients with coronary artery disease and low ejection fraction. Hellenic Journal of Cardiology 2009;50:45-51.
13. Sadeghian H, Majd-Ardakani J, Lotfi-Tokaldany M. Assessment of Myocardial Viability: Selection of Patients for Viability Study and Revascularization. Journal of Tehran University Heart Center 2009; 4:5-15.
14. Sun JP, Popović ZB, Greenberg NL, Xu XF, Asher CR, Stewart WJ, et al. Noninvasive quantification of regional myocardial function using Doppler-derived velocity, displacement, strain rate, and strain in healthy volunteers: effects of aging. J Am Soc Echocardiography 2004; 17:132-8.
15. Palmes PP, Masuyama T, Yamamoto K, Kondo H, SakataY, Takiuchi S, et al. Myocardial longitudinal motion by tissue velocity imaging in the evaluation of patients with myocardial infarction. J Am Soc Echocardiography 2000;13: 818–26.
16. Dagianti A, Vitarelli A, Conde Y, Penco M, Fedele F, Dagianti A. Assessment of regional left ventricular func-

tion during exercise test with pulsed tissue Doppler imaging. Am J Cardiology 2000; 86(suppl): 30G-32G.
17. Naqvi TZ, Nevman G, Broyde A, and siegel RJ. Myocardial Doppler tissue imaging: findings in inferior myocardial infarction and left ventricular hypertrophy-wall motion assessment. J Am Soc Echocardiography. 2001; 14:864-73.
18. Weidemann F, Dommke C, Bijnens B, Claus P, D'hooge J, Mertens P, et al. Defining the transmurality of a chronic myocardial infarction by ultrasonic strain-rate imaging: implications for identifying intramural viability: an experimental study. Circulation 2003;107: 883-8
19. Hoffmann R, Altiok E, Nowak B, Heussen N, Ku¨hl H, Kaiser HJ, et al. Strain Rate Measurement by Doppler Echocardiography Allows Improved Assessment of Myocardial Viability in Patients With Depressed Left Ventricular Function. J Am Coll Cardiology 2002; 39:443–9.
20. Cain P, Khoury V, Short L, Marwick TH. Usefulness of quantitative echocardiographic techniques to predict recovery of regional and global left ventricular function after acute myocardial infarction. American Journal of Cardiology. 2003;91:391-6.
21. Marwick TH. Measurement of strain and strain rate by echocardiography: ready for prime time? J Am Coll CArdiol 2006; 47: 1313-27.