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CHAPTER 9

Longitudinal mechanics of the periinfarct zone and ventricular tachycardia inducibility in patients with chronic ischemic cardiomyopathy.

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Background. Quantification of segmental left ventricular (LV) strain by speckle-tracking echocardiography can identify transmural infarcts in patients with chronic ischemic cardiomyopathy. The aim of the study was to explore the relationship between the LV longitudinal peak systolic strain (LPSS) of the infarct, peri-infarct and remote zones and monomorphic ventricular tachycardia (VT) inducibility on electrophysiological (EP) study.

Methods. A total of 134 patients with chronic ischemic cardiomyopathy scheduled for EP study were included. The protocol consisted of clinical, ECG and echocardiographic evaluation, including LV longitudinal strain analysis using speckle-tracking echocardiography, immediately before EP study. An infarct segment was defined as a longitudinal strain value of >-5%, and a peri-infarct segment was defined as immediately adjacent to an infarct segment.

Results. The infarct zone had the most impaired longitudinal strain (-0.5±3.0%), whereas the peri-infarct and remote zones had more preserved longitudinal strain (-10.8±1.9% and -14.5±3.0%, respectively; ANOVA p<0.001). Seventy-two (54%) patients had inducible monomorphic VT on EP study. There was no significant difference in LVEF (31±9% vs. 32±11%, p=0.29) between inducible and non-inducible patients. LPSS of the peri-infarct zone was more impaired in inducible patients (-9.8±1.5% vs. -11.0±2.1%, p=0.001), but no differences in LPSS of the infarct (-0.5±3.2% vs. -0.4±2.7%, p=0.75) and remote (-14.6±2.8% vs. -14.5±3.4%, p=0.92) zones were observed. Only LPSS of the peri-infarct zone (OR 1.43, 95% CI 1.15-1.78, p=0.001) was independently related to monomorphic VT inducibility on multiple logistic regression.

Conclusions. Longitudinal strain analysis may be a useful imaging tool to risk-stratify ischemic patients for malignant ventricular arrhythmia.
INTRODUCTION

Coronary artery disease, provoking lethal ventricular arrhythmias, is one of the most common causes of sudden cardiac death. Usually, the electrical sequence is due to the initial development of monomorphic ventricular tachycardia (VT) that subsequently degenerate into ventricular fibrillation. The classical anatomical substrate for monomorphic VT is the peri-infarct zone with re-entry pathways that abounds the infarcted myocardial scar tissue. These scar areas represented by the infarct zone are normally highly fibrosed, whereas the peri-infarct zone constitutes a highly heterogeneous area with intermediate degrees of non-transmural fibrosis. The tissue heterogeneity of the peri-infarct zone may be electrically unstable and constitute the substrate for re-entrant VT.

Recently cardiac magnetic resonance imaging (CMR) permits identification of myocardial scar tissue and can also characterize the peri-infarct zone. Specifically, the extent of peri-infarct zone can be quantified with contrast-enhanced CMR, whereas the mechanical properties of the peri-infarct zone can be assessed by tagged CMR. These studies demonstrated a strong relationship between the peri-infarct zone heterogeneity and monomorphic VT inducibility.

Novel speckle-tracking echocardiography with quantifications of regional left ventricular (LV) longitudinal strain has been shown to have good sensitivity and specificity in identifying transmural scar tissue by contrast-enhanced CMR. Therefore, regional speckle-tracking analysis of LV longitudinal peak systolic strain (LPSS) permits the differentiation of the infarct, peri-infarct and remote zones by characterizing the different tissue mechanical properties. Thus, the aim of the present study was to explore the relationship between the LPSS of the peri-infarct zone detected with speckle-tracking echocardiography and monomorphic VT inducibility in patients with chronic ischemic cardiomyopathy.

METHODS

Patient population and protocol

A total of 141 consecutive patients scheduled for cardiac electrophysiological (EP) study were included. Inclusion criteria were previous history of myocardial infarction (>40 days ago), referral for a clinically indicated EP study because of syncope or non-sustained ventricular tachycardia at ECG-Holter monitoring, and sinus rhythm during the echocardiographic examination.
The protocol consisted of an extensive clinical, electrocardiographic (ECG), and echocardiographic evaluation, including LPSS analysis. Afterwards (maximum 24 hours later), all patients underwent a clinically indicated EP study to induce monomorphic VT. All echocardiographic analyses were performed by an independent observer blinded to the EP study results. The echocardiographic analyses included assessments of LV volumes, LV ejection fraction (LVEF) and wall motion scoring. Speckle-tracking analysis using automated function imaging was applied to determine global LPSS. In addition, the LPSS of the infarct, peri-infarct and remote zones were determined based on segmental strain values. Finally, the clinical, ECG, and echocardiographic variables were tested by uni- and multivariable logistic regression analysis to investigate the association with inducibility of monomorphic VT at EP study.

**Standard echocardiography**

All patients were imaged in left lateral decubitus position using a commercially available system (Vingmed Vivid 7, General Electric-Vingmed, Milwaukee, Wisconsin, USA). Standard 2-dimensional images were obtained using a 3.5-MHz transducer and digitally stored in cine-loop format; the analysis was performed offline using EchoPAC version 108.1.5 (General Electric-Vingmed).

LV end-diastolic volume (LVEDV), end-systolic volume (LVESV) and LVEF were calculated using Simpson’s biplane method of discs as recommended by the American Society of Echocardiography guidelines.

Segmental wall motions were evaluated and scored as 1: normal; 2: hypokinesia; 3: akinesia; 4: dyskinesia. Global wall motion score index was calculated using the sum of the segmental scores divided by the number of segments analyzed, as previously described.

**Speckle-tracking analysis**

The speckle-tracking software tracks frame-to-frame movements of natural myocardial acoustic markers, or speckles, on standard gray scale images. Each region of the myocardium has a characteristic speckle-pattern; therefore it can be followed during the cardiac cycle. Furthermore, speckle-tracking analysis is an angle independent technique that allows the evaluation of myocardial contraction/relaxation in the circumferential, longitudinal and radial directions.
Myocardial strain can be assessed from temporal differences in the mutual distance of neighboring speckles. The change in length/initial length of the speckle-pattern over the cardiac cycle can be used to calculate longitudinal strain, with myocardial shortening represented as negative strain, and myocardial lengthening as positive strain. During the systole the myocardium shortens in the longitudinal direction and, conventionally, is presented as negative values. Therefore, more negative values indicate larger and more preserved longitudinal strain. LV longitudinal strain was determined from the apical long-axis, 4- and 2-chamber views with optimal 2D gray scale image frame rates ranging from 50 to 100 frame/s. After defining the mitral annulus and the LV apex with 3 index points at the end-systolic frame in each apical view, the automated function imaging software automatically traces the endocardial border while allowing the user to manually adjust the region of interest width to include the entire myocardial wall. Further manual adjustments of the endocardial border were performed to ensure adequate tracking throughout the cardiac cycle. The LV was automatically divided in 6 segments in each apical view and tracking quality was validated for each segment. Finally, the automated algorithm provides the LPSS value for each LV segment in a 17-segment model polar plot, with the average value of LPSS for each apical view and the averaged global LPSS value for the entire LV.

According to previous evidence, a segmental longitudinal strain value >-5% was consistent with transmural scar on delayed enhancement cardiac magnetic resonance imaging, and was classified as infarcted segment. Thus, all segments adjacent to the infarcted segments were classified as peri-infarct segments and all remaining segments were classified as remote segments (Figure 1). Finally, LPSS of the infarct, peri-infarct and remote zones were calculated as the means of the LPSS of the infarct, peri-infarct and remote segments respectively.

The intra- and inter-observer variabilities for longitudinal strain analysis as assessed with Bland-Altman analysis were previously reported and were -0.3 ± 0.6% and -0.2 ± 2.6%, respectively.

**Cardiac electrophysiological testing**

The cardiac EP study was performed within 24 hours after echocardiographic evaluations. Studies were performed in the post absorptive, non-sedated state. Antiarrhythmic drugs were discontinued for 5 half-lives, with the exception of amiodarone which was continued in 13 patients.
The EP study consisted of a programmed extrastimulation protocol using 3 drive cycle lengths (600, 500, 400 ms) and 1, 2 and 3 extrastimuli while pacing at 2 right ventricular sites (right ventricular apex and right ventricular outflow tract). The initial S2 coupling interval in each stimulation sequence was at least 300 ms and stimulation was not performed at a coupling interval of <200 ms. Inducibility at EP study was defined as the induction of a monomorphic VT lasting for more than 30 seconds or monomorphic VT requiring termination because of hemodynamic compromise.

**Statistical analysis**

All continuous variables are presented as mean ± SD. Categorical data are presented as numbers and percentages. Differences in LPSS and number of segments between the different zones (infarct, peri-infarct and remote) in the overall population were assessed by one-way analysis of variance (ANOVA), and post-hoc analyses were performed with Bonferroni correction. Unpaired Student’s t-test and Chi-square test were used to compare continuous and categorical variables respectively for inducible
vs. non-inducible patients. In order to identify variables related to a positive response to EP study, uni- and multivariable logistic regression analyses were performed and included clinical (age, gender, etiology, cardiovascular risk factors, time since last myocardial infarction, New York Heart Association [NYHA] functional class, heart rate, and medications), ECG (PR, QRS duration and corrected QT interval duration), and echocardiographic (LV volumes, LVEF, wall motion score index, global LPSS, infarct LPSS, peri-infarct LPSS, remote LPSS) variables. Only univariable predictors with a p value ≤0.10 were entered as covariates in multivariable logistic regression model using an enter method. Odds ratio and 95% confidence intervals were calculated.

All statistical tests were 2-sided, and a p value <0.05 was considered significant. A statistical software program SPSS 16.0 (SPSS Inc, Chicago, IL, USA) was used for statistical analysis.

No extramural funding was used to support this work. The authors are solely responsible for the design and conduct of this study, all study analyses, the drafting and editing of the paper and its final contents.

RESULTS

Overall population

Reliable speckle-tracking was obtained in 134 (95%) patients and constituted the final study population. Tables 1 and 2 show the demographic and clinical characteristics of the overall patient population. In particular, the mean age of the overall patient population was 67 ± 10 years and 119 (89%) patients were men. Both the QRS duration and the corrected QT interval were slightly prolonged (122 ± 28 ms and 444 ± 35 ms respectively). Beta-blockers and angiotensin-converting enzyme inhibitor or angiotensin receptor blockers treatments were present in 67% and 92% of the patients, respectively; whereas 10% and 16% of the patients were treated with amiodarone or sotalol, respectively.

All the patients had dilated LV volumes (LVEDV and LVESV were 171 ± 57 ml and 120 ± 50 ml respectively) and reduced LVEF (31 ± 10%). The mean global LPSS was -9.1 ± 3.8%. A progressive increment in LPSS from the infarct to peri-infarct and remote zones (ANOVA p <0.001; Figure 2) was observed. The infarct zone showed the most impaired LPSS values (-0.5 ± 3.0%), whereas the peri-infarct and remote zones showed more preserved values of LPSS (-10.8 ± 1.9% and -14.5 ± 3.0%, respectively; Figure 2).
The EP study was conducted in all the patients without major complications. After EP study, 72 (54%) patients were classified as inducible and 62 (46%) as non-inducible. Table 1 shows the demographic and clinical characteristics of inducible and non-inducible patients.

**Table 1.** Demographic and clinical characteristics of overall population, and inducible vs. non-inducible patients.

<table>
<thead>
<tr>
<th></th>
<th>Overall population (n=134)</th>
<th>Inducible monomorphic VT (n=72)</th>
<th>Non-inducible monomorphic VT (n=62)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yrs)</td>
<td>67±10</td>
<td>67±10</td>
<td>67±10</td>
<td>0.84</td>
</tr>
<tr>
<td>Male (%)</td>
<td>119 (89)</td>
<td>67 (87)</td>
<td>52 (90)</td>
<td>0.093</td>
</tr>
<tr>
<td>Hypertension (%)</td>
<td>58 (43)</td>
<td>31 (43)</td>
<td>27 (44)</td>
<td>0.95</td>
</tr>
<tr>
<td>Diabetes (%)</td>
<td>35 (26)</td>
<td>17 (24)</td>
<td>18 (29)</td>
<td>0.47</td>
</tr>
<tr>
<td>Family history of cardiac disease (%)</td>
<td>46 (34)</td>
<td>29 (40)</td>
<td>17 (27)</td>
<td>0.12</td>
</tr>
<tr>
<td>Current smoker (%)</td>
<td>21 (16)</td>
<td>11 (15)</td>
<td>10 (16)</td>
<td>0.89</td>
</tr>
<tr>
<td>Time since last myocardial infarction (months)</td>
<td>88±90</td>
<td>100±94</td>
<td>74±83</td>
<td>0.090</td>
</tr>
<tr>
<td>NYHA functional class III-IV (%)</td>
<td>47 (35)</td>
<td>24 (33)</td>
<td>23 (37)</td>
<td>0.65</td>
</tr>
<tr>
<td>Heart rate (bpm)</td>
<td>67±10</td>
<td>66±11</td>
<td>68±10</td>
<td>0.13</td>
</tr>
<tr>
<td>PR interval (ms)</td>
<td>182±32</td>
<td>185±32</td>
<td>178±33</td>
<td>0.21</td>
</tr>
<tr>
<td>QRS duration (ms)</td>
<td>122±28</td>
<td>126±27</td>
<td>117±28</td>
<td>0.044</td>
</tr>
<tr>
<td>Corrected QT interval duration (ms)</td>
<td>444±35</td>
<td>446±33</td>
<td>441±36</td>
<td>0.45</td>
</tr>
</tbody>
</table>


**Figure 2.** Differences in longitudinal peak systolic strain (LPSS) among infarct, peri-infarct and remote zones.
inducible patients. There were no significant differences in age, gender, cardiac risk factors, NYHA functional class and heart rate. Inducible patients tended to have longer time since last myocardial infarction (100 ± 94 months vs. 74 ± 83 months, p = 0.090). In addition, QRS duration was significantly longer in inducible patients (126 ± 27 ms vs. 117 ± 28 ms, respectively, p = 0.044). Table 2 showed no significant differences in the use of cardiac medication between inducible and non-inducible patients.

In regards to the echocardiographic variables, there was a trend towards a more dilated LV in inducible patients (178 ± 60 ml vs. 161 ± 53 ml, p = 0.094) but there were no significant differences in LVEF (31 ± 9% vs. 32 ± 11%, p = 0.29). However, LPSS of the peri-infarct zone was more impaired in inducible patients (-9.8 ± 1.5% vs. -11.0 ± 2.1%, respectively, p = 0.001), but there were no significant differences in LPSS of the infarct (-0.5 ± 3.2% vs. -0.4 ± 2.7%, p = 0.75) and remote (-14.6 ± 2.8% vs. -14.5 ± 3.4%, p = 0.92) zones (Table 3). Figure 3 shows examples of patients with and without inducible monomorphic VT. The patient with inducible monomorphic VT had a more reduced LPSS of the peri-infarct zone. Furthermore, in this subgroup of patients mean VT cycle length was 288 ± 65 ms and this was modestly but significantly related to global LPSS (r = 0.52, p <0.001; Figure 4). Conversely, VT cycle length was not correlated to LPSS of the peri-infarct zone.

Table 2. Medications use of overall population, and inducible vs. non-inducible patients.

<table>
<thead>
<tr>
<th>Medications</th>
<th>Overall population (n=134)</th>
<th>Inducible monomorphic VT (n=72)</th>
<th>Non-inducible monomorphic VT (n=62)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Beta-blockers (%)</td>
<td>90 (67)</td>
<td>46 (64)</td>
<td>44 (71)</td>
<td>0.38</td>
</tr>
<tr>
<td>ACE inhibitor/Angiotensin receptor blockers (%)</td>
<td>124 (92)</td>
<td>64 (89)</td>
<td>60 (97)</td>
<td>0.083</td>
</tr>
<tr>
<td>Ca-antagonists (%)</td>
<td>12 (9)</td>
<td>9 (12)</td>
<td>3 (5)</td>
<td>0.12</td>
</tr>
<tr>
<td>Sotalol (%)</td>
<td>21 (16)</td>
<td>12 (17)</td>
<td>9 (15)</td>
<td>0.73</td>
</tr>
<tr>
<td>Amiodarone (%)</td>
<td>13 (10)</td>
<td>6 (8)</td>
<td>7 (11)</td>
<td>0.56</td>
</tr>
<tr>
<td>Diuretics (%)</td>
<td>90 (67)</td>
<td>49 (68)</td>
<td>41 (66)</td>
<td>0.81</td>
</tr>
<tr>
<td>Nitrates (%)</td>
<td>22 (16)</td>
<td>14 (19)</td>
<td>8 (13)</td>
<td>0.31</td>
</tr>
<tr>
<td>Statins (%)</td>
<td>106 (79)</td>
<td>56 (78)</td>
<td>50 (81)</td>
<td>0.68</td>
</tr>
<tr>
<td>Acetil salicylic acid/anticoagulants (%)</td>
<td>126 (94)</td>
<td>68 (94)</td>
<td>58 (94)</td>
<td>0.83</td>
</tr>
</tbody>
</table>

ACE: angiotensin-converting enzyme; MVT: monomorphic ventricular tachycardia
After analyzing all clinical, ECG and echocardiographic variables, male gender, time since last myocardial infarction, QRS duration, angiotensin-converting enzyme inhibitor or angiotensin receptor blockers usage, LVEDV and LPSS of the peri-infarct zone had a p value ≤0.10 on univariable logistic regression analysis and were entered in the multivariable model (Table 4). Only LPSS of the peri-infarct zone (odds ratio 1.43, 95% confidence intervals 1.15-1.78, p = 0.001) was independently related to monomorphic VT inducibility on EP study.

**DISCUSSION**

The current study showed that in patients with chronic ischemic cardiomyopathy, LPSS of the peri-infarct zone was strongly associated to monomorphic VT inducibility. In particular, after correction for clinical, ECG and echocardiographic variables, LPSS of the peri-infarct zone was the only independent factor related to monomorphic VT inducibility.

<table>
<thead>
<tr>
<th>Overall population (n=134)</th>
<th>Inducible monomorphic VT (n=72)</th>
<th>Non-inducible monomorphic VT (n=62)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>LVEDV (ml)</td>
<td>171±57</td>
<td>178±60</td>
<td>161±53</td>
</tr>
<tr>
<td>LVESV (ml)</td>
<td>120±50</td>
<td>126±52</td>
<td>113±47</td>
</tr>
<tr>
<td>LVEF (%)</td>
<td>31±10</td>
<td>31±9</td>
<td>32±11</td>
</tr>
<tr>
<td>WMSI</td>
<td>1.7±0.4</td>
<td>1.8±0.4</td>
<td>1.7±0.4</td>
</tr>
<tr>
<td>Global LPSS (%)</td>
<td>-9.1±3.8</td>
<td>-9.1±3.4</td>
<td>-9.0±4.2</td>
</tr>
<tr>
<td>LPSS of the infarct zone (%)</td>
<td>-0.5±3.0</td>
<td>-0.5±3.2</td>
<td>-0.4±2.7</td>
</tr>
<tr>
<td>LPSS of the peri-infarct zone (%)</td>
<td>-10.8±1.9</td>
<td>-9.8±1.5</td>
<td>-11.0±2.1</td>
</tr>
<tr>
<td>LPSS of the remote zone (%)</td>
<td>-14.5±3.0</td>
<td>-14.6±2.8</td>
<td>-14.5±3.4</td>
</tr>
</tbody>
</table>

LPSS: longitudinal peak systolic strain; LVEDV: left ventricular end-diastolic volume; LVEF: left ventricular ejection fraction; LVESV: left ventricular end-systolic volume; MVT: monomorphic ventricular tachycardia; WMSI: wall motion score index.
Figure 3. Example of 2 patients with and without inducible monomorphic ventricular tachycardia (VT). The panels show the polar plot model illustrating the values of the longitudinal peak systolic strain for every segment (left) and the electrocardiogram at 100 mm/sec obtained during the electrophysiological test for inducibility (right). Panel A. Patient with postero-lateral scar with inducible monomorphic VT. LPSS of the peri-infarct zone was -9.0%. Panel B. Patient with septal and apical scar without inducible monomorphic VT. LPSS of the peri-infarct zone was more preserved (-11.0%) as compared to the inducible patient.

Figure 4. Panel A: Correlation between ventricular tachycardia (VT) cycle length and global longitudinal peak systolic strain (LPSS). Panel B: Correlation between ventricular tachycardia (VT) cycle length and LPSS of the peri-infarct zone.
Peri-infarct zone and monomorphic VT

In the chronic phase of myocardial infarction, monomorphic VT is caused by re-entrant pathways around the borders of infarcted scar area. Therefore, the peri-infarct zone is the anatomical substrate for these malignant ventricular arrhythmias.

Previous animal studies demonstrated that 2 months after induction of myocardial infarction, the myofibers of the peri-infarct zone were highly disorganized with replacement by connective tissue. In particular, peri-infarct zone was characterized by non-uniform anisotropic areas with isles of viable myocardium and fibrosis, resulting in slow electrical conduction of the cardiac pulse. This tissue heterogeneity with altered mechanical properties results in electrical instability and may predispose to ventricular arrhythmias.

The tissue heterogeneity of the peri-infarct zone has been investigated with different techniques trying to characterize the electrical, anatomical and mechanical properties.

The electrical characterization of the peri-infarct zone has been studied with bipolar electrograms derived from electro-anatomic mapping techniques. The infarct areas had the lowest voltage amplitudes whereas the remote areas had the highest voltages amplitudes. The peri-infarct zone had intermediate voltage amplitudes and subsequently, several clinical studies have demonstrated that these areas were the target of successful monomorphic VT ablation. Recently, contrast-enhanced
CMR has been shown to be a useful imaging technique to anatomically characterize the peri-infarct zone.\textsuperscript{5, 17, 18} Whereas the infarct zone shows high-intensity contrast signal, the peri-infarct zone shows an intermediate-intensity contrast signal that can be quantified with dedicated algorithms.\textsuperscript{8, 17, 18} The extent of the peri-infarct zone has been related to an increased mortality after myocardial infarction, and increased inducibility for monomorphic VT on EP testing.\textsuperscript{8, 18} Indeed, Yan et al.\textsuperscript{18} demonstrated that the extent of peri-infarct zone predicted an increased mortality in patients with previous myocardial infarction. Subsequently, Schmidt et al.\textsuperscript{8} found that that larger extent of peri-infarct zone was independently related with an increased inducibility for sustained monomorphic VT. More recently, Roes et al.\textsuperscript{17} corroborated these previous observations by reporting the association between the peri-infarct zone extent and the occurrence of spontaneous ventricular arrhythmia detected as appropriate therapies of implantable cardioverter-defibrillator (ICD) in 91 patients with previous myocardial infarction and who underwent ICD implantation.

Finally, the mechanical properties of the peri-infarct zone have been recently studied by Fernandes et al.\textsuperscript{7} using tagged CMR. The authors evaluated the circumferential strain and time to peak circumferential strain at the peri-infarct zone. An enhanced peak circumferential strain and earlier time to peak circumferential strain at the peri-infarct zone were positively related with monomorphic VT inducibility.

In the present study 2-dimensional speckle-tracking analyses of the standard grey-scale echocardiographic images was used. This imaging technique provides valuable information on the mechanical properties of the peri-infarct zone. Particularly, in the current study the longitudinal mechanics of the peri-infarct zone was investigated. Inducible patients had larger impairment of LPSS in the peri-infarct zone (-9.8 ± 1.5\% vs. -11.0 ± 2.1\%, \textit{p} = 0.001). Furthermore, after correcting for clinical, ECG and echocardiographic variables, LPSS of the peri-infarct zone was the only independent determinant of monomorphic VT inducibility (adjusted odds ratio 1.39, 95\% confidence interval 1.11-1.78, \textit{p} = 0.005). Therefore, this observation confirmed the independent relationship between the mechanical properties of the peri-infarct zone, particularly the longitudinal mechanics, and monomorphic VT inducibility.

Importantly, in contrast to global LPSS, peri-infarct peak longitudinal strain was not related to VT cycle length. This finding underscores that the LPSS values of peri-infarct zone did not reflect the extension of the scar but only the longitudinal function of the tissue adjacent to the infarcted segments. Conversely, global LPSS may reflect the total scar burden,\textsuperscript{19} and more impaired global LPSS are associated to longer VT cycle length.
Clinical implications

The present study underscores the independent relationship between reduced peri-infarct zone longitudinal strain and VT inducibility in patients with chronic ischemic cardiomyopathy. Regional LV longitudinal strain analysis by speckle-tracking echocardiography may be helpful in identifying patients with increased risk of malignant ventricular arrhythmias. Furthermore, quantifications of LPSS of the peri-infarct zone may be a potentially useful tool for selecting patients who might benefit from ICD therapy.

CONCLUSIONS

In patients with chronic ischemic cardiomyopathy, LPSS of the peri-infarct zone was independently associated to monomorphic VT inducibility. Thus, speckle-tracking analysis may be a useful imaging tool to risk-stratify patients with chronic ischemic cardiomyopathy who might benefit from ICD.
Longitudinal mechanics of the periinfarct zone and ventricular tachycardia inducibility in patients with cardiomyopathy

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Chapter 9


