Two-dimensional strain technique to detect the function of coronary collateral circulation
Jingjing Yang, Xiaoling Liu, Guihua Jiang, Yuguo Chen, Yun Zhang and Mei Zhang

Background We aimed to evaluate the function of coronary collateral circulation (CCC) in the ischemic myocardium by a two-dimensional strain technique.

Methods and results Myocardial segments for all patients were divided into three groups: no coronary collateral circulation (NCCC), CCC, and normal control. We obtained the segmental strain and the strain rate in longitudinal, radial, circumferential directions, and peak rotation and rotation rate of the left ventricle by ultrasonography. Also, the time to peak of all the strain and the strain rate were acquired. The peak systolic strain, the end systolic strain, the peak systolic strain rate in the longitudinal direction, the peak systolic strain rate in the radial direction, the peak systolic strain, the end systolic strain, the peak systolic strain rate, the peak early diastolic strain rate in the circumferential direction, the peak systolic rotation rate, and the peak early diastolic rotation rate were larger for the CCC group than the NCCC group (P<0.05). Also, the time to peak of the segments was delayed for the NCCC group than for the CCC group. Receiver operating characteristic curve analysis was performed for the late diastolic strain rate in the circumferential direction to predict the presence of CCC.

Conclusion CCC could partially maintain the function of the related ischemic myocardium segments and prevent mechanical dyssynchrony. The two-dimensional strain technique is convenient and noninvasive for predicting the presence of CCC. Coron Artery Dis 00: 000–000 © 2012 Wolters Kluwer Health | Lippincott Williams & Wilkins.

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Keywords: coronary artery, echocardiography, myocardium ischemia, strain rate, two-dimensional strain

Introduction
Coronary artery disease (CAD) is the leading cause of death worldwide. Reperfusing blood to the ischemic heart is the traditional therapy, and many revascularization procedures have been devised. However, 20–30% of patients with CAD cannot undergo percutaneous coronary intervention or coronary artery bypass grafting surgery because of severe coronary atherosclerosis [1]. Controlling symptoms and altering the course of advanced CAD may be an alternative therapy by inducing natural bypass therapies. Promoting coronary collateral growth can improve blood supply to a myocardial area jeopardized by ischemia [2] and could be a substitute for patients with severe coronary atherosclerosis. However, the use of coronary collaterals has been controversial, probably because of the endpoints examined (mitigating myocardial infarcts, preventing the formation of left-ventricular aneurysm and improving survival), and these processes are indirect. Two-dimensional (2-D) strain may be used to examine the systolic and diastolic function of the ischemic myocardium directly and noninvasively.

A 2-D strain technique based on speckle tracking offers advantages of angle independence over the Doppler-derived approach: high sensitivity to detect myocardium dysfunction early, noninvasiveness, and convenience. Assessments of myocardial function derived by speckle tracking have been validated in animal and human studies [3–5]. However, few studies have investigated the significant role of this technique in evaluating the function of the coronary collateral artery in the ischemic myocardium and predicting the presence of the coronary collateral artery.

We used 2-D strain to verify the effectiveness of coronary collateral circulation (CCC) in ischemic myocardium function and mechanical dyssynchrony in patients.

Methods
Study population
All consecutive patients with unstable angina pectoris who were undergoing coronary angiography (CAG) in our hospital between September 2007 and May 2008 were eligible for this study. Exclusion criteria were acute myocardial infarction, valvular cardiac disease, cardiomyopathy, severe heart failure (New York Heart Association class IV), and arrhythmia.

Cardiac ultrasound examination
Echocardiography was performed within 24 h before CAG using a commercially available ultrasound system (M8 probe, Vivid 7; GE Medical Systems; Horten, Norway). Ultrasonography with standard parasternal and apical...
views was performed in patients in the left lateral recumbent position. We obtained high-quality 2-D images including apical (four-chamber, two-chamber, and long axis) and short-axis views (mitral annulus, papillary muscle, and apex) using a 2.0–4.0 MHz transducer at a frame rate of 60–100 frames/s and three consecutive cardiac cycles during breath hold. Images were digitized in a cine-loop format and stored.

**Coronary artery angiography**

Standard selective CAG with at least four views of the left coronary system and two views of the right coronary artery (RCA) were performed by the Judkins technique. Coronary angiograms were examined for collateral development by two experienced interventional cardiologists from our institute who were blinded to the study. Differences in interpretation were adjudicated by a third investigator.

A 17-segment left-ventricle model recommended by the American Society of Echocardiography was used to analyze the regional myocardial function. Every segment was grouped into the left anterior descending (LAD) territory, the left circumflex (LCX) territory, and the RCA territory on the basis of known vascular distribution. Then, combining the results of CAG and the corresponding blood supply of myocardial segments, patients were divided into the following three groups: no coronary collateral circulation (NCCC) – segments in the territory as one of the three main arteries (RCA, LAD branch, and LCX branch) showed stenosis at least 75% but no CCC; CCC – segments in the territory as one of the three main arteries showed stenosis at least 75% and CCC; and normal control – segments in the same territory with basically normal coronary circulation on the basis of CAG. The study protocol was approved by the local ethics committee, and written informed consent was obtained from all participants.

**Echocardiography analysis**

Echocardiography images were analyzed offline using EchoPAC PC SW-Only v.7.0.x (GE-Vingmed, Horten, Norway). The software can divide every view of the left-ventricular walls (four-chamber, two-chamber, and long-axis views, and mitral annulus, papillary muscle, and apex views) into six segments automatically. Then, strain, strain rate, and time to peak of the values of the myocardial segments were acquired using the software and analyzed using Microsoft Excel.

The following parameters of the myocardial segments were calculated: the systolic peak strain (Sp), the systolic peak strain rate (Sr), the end systolic strain (Se), the peak early diastolic strain rate (Ser), and the peak late diastolic strain rate (Sra) in longitudinal (L), radial (R), circumferential (C) directions, and the peak systolic rotation (Rot), the peak systolic Rot rate, the peak early diastolic Rot rate, and the peak late diastolic Rot rate. Time to peak strain and strain rate were measured.

**Statistical analysis**

All numerical data in normal distribution were analyzed by one-sample Kolmogorov–Smirnov test and expressed as mean ± SD. Comparison of continuous variables between two groups involved an unpaired t-test and comparison among multiple groups analysis of variance. Receiver operating characteristic curve (ROC) analysis was carried out to identify the sensitivity and the specificity of strain parameters for predicting the presence of CCC. Data analysis involved the use of SPSS v13.0 (SPSS Inc., Chicago, Illinois, USA). A P-value less than 0.05 was considered statistically significant.

**Results**

**Clinical characteristics**

We enrolled 118 patients (mean age 55 ± 7 years; 71% males). The mean history of unstable angina pectoris in all patients was 6.7 ± 6.9 years. We detected and recorded baseline and risk factors, including diabetes, history of smoking and levels of total cholesterol, triglycerides, low-density lipoprotein cholesterol, and high-density lipoprotein cholesterol. The presence of diabetes mellitus was defined as a fasting plasma glucose 126 mg/dl or more and/or a 2 h post-glucose load 200 mg/dl or more or the use of specific antidiabetic drug therapy. The clinical data and the results of statistical analysis are shown in Table 1. The three groups did not differ in age, sex, smoking, or clinical risk factors, including blood pressure, lipid levels, or fasting blood glucose.

<table>
<thead>
<tr>
<th>Variables</th>
<th>Normal controls</th>
<th>CCC</th>
<th>NCCC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>56.38 ± 8.33</td>
<td>56.7 ± 8.54</td>
<td>57.2 ± 9.97</td>
</tr>
<tr>
<td>Diabetes (%)</td>
<td>20 (66.7%)</td>
<td>34 (85%)</td>
<td>30 (80%)</td>
</tr>
<tr>
<td>History of smoking (%)</td>
<td>10 (33.3%)</td>
<td>20 (50%)</td>
<td>22 (58%)</td>
</tr>
<tr>
<td>Systolic blood pressure (mmHg)</td>
<td>150.50 ± 24.59</td>
<td>148 ± 24.55</td>
<td>138 ± 23.71</td>
</tr>
<tr>
<td>Diastolic blood pressure (mmHg)</td>
<td>45.87 ± 45.15</td>
<td>57.31 ± 42.61</td>
<td>50.20 ± 43.45</td>
</tr>
<tr>
<td>Total cholesterol (mmol/l)</td>
<td>4.83 ± 1.23</td>
<td>4.55 ± 1.05</td>
<td>4.75 ± 1.23</td>
</tr>
<tr>
<td>Triglyceride level (mmol/l)</td>
<td>1.78 ± 1.35</td>
<td>1.80 ± 0.90</td>
<td>1.60 ± 0.75</td>
</tr>
<tr>
<td>Low-density lipoprotein cholesterol (mmol/l)</td>
<td>2.70 ± 1.47</td>
<td>2.53 ± 1.28</td>
<td>2.71 ± 1.38</td>
</tr>
<tr>
<td>High-density lipoprotein cholesterol (mmol/l)</td>
<td>1.22 ± 0.45</td>
<td>0.93 ± 0.42</td>
<td>1.02 ± 0.33</td>
</tr>
<tr>
<td>Fasting blood glucose (mmol/l)</td>
<td>5.45 ± 1.21</td>
<td>5.99 ± 1.83</td>
<td>6.41 ± 2.34</td>
</tr>
</tbody>
</table>

Data are mean ± SD or number (%) of patient. Normal control group, same segments in the territory with artery stenosis less than 20% on the basis of coronary angiography. CCC, coronary collateral circulation (CCC, segments in the territory with one of the three main arteries with stenosis); NCCC, no coronary collateral circulation (NCCC, segments in the territory with one of the three main arteries including right coronary artery, left anterior descending branch, and left circumflex branch stenosis).
Coronary angiography
Among all patients, we found 167 coronary arteries with stenosis at least 75%. There were 48 coronary collaterals: 14 branches from the LAD to the RCA, 18 branches from the RCA to the LAD, five branches from the RCA to the LCX, seven branches from the LCX to the RCA, two branches from the LCX to the LAD, and two branches from the LAD to the LCX (Fig. 1).

All the collaterals were where the diameter showed stenosis 85% or more in this study.

Myocardial segments to analysis
We analyzed 974 myocardial segments in total. We found 226, 152, and 155 segments in the long axis for the NCCC, CCC, and normal groups, respectively, and 216, 110, and 115 segments, respectively, in the short axis.

Function of coronary collateral circulation
During systole and diastole, the myocardium exhibits negative and positive strain and strain rates in different directions. Because the peak strain and strain rate represents the highest extent of myocardium deformation during the cardiac cycle, which may be positive or negative in different directions, we compared only the absolute value of the variable.

Sps and Ses were significantly larger for the CCC than the NCCC group in the longitudinal and the circumferential directions (LSps, \(P = 0.003\); LSes, \(P = 0.001\); CSps, \(P = 0.007\); CSes, \(P = 0.001\)); the time to peak strain was significantly delayed for the NCCC than the CCC group (TLSps, \(P = 0.001\); TLSes, \(P = 0.024\); TCSps, \(P = 0.000\); TCSes, \(P = 0.000\)) (Table 2).

The normal and the CCC groups did not differ in the time to peak diastolic strain rate (TSRe, \(P = 0.196\); TSRa, \(P = 0.381\)). The peak systolic strain rate was larger for the CCC than the NCCC group (LSRs, \(P = 0.006\); CSRs, \(P = 0.013\)); hence, the myocardial segments with CCC have better function than those without CCC, especially systolic function, in the longitudinal and circumferential directions (Figs 2 and 3).

The strain rate (SRs, \(P = 0.000\); SRe, \(P = 0.679\); and Sra, \(P = 0.067\)) in the radial direction and the Rot rate for the three groups are shown in Table 3. The strain rate was better for the CCC than the NCCC group (SRs, \(P = 0.000\); SRe, \(P = 0.000\)) and the CCC and the NCCC groups did not differ in the peak time to diastolic strain rate (SRe, \(P = 0.196\); Sra, \(P = 0.381\)). Compared with the NCCC group, the CCC group had a higher Rot rate (RotR Peak S, \(P = 0.018\); RotR Peak E, \(P = 0.003\)). Compared with the CCC group, the NCCC group showed delayed time to RotR Peak S and RotR Peak E (\(P = 0.000\) and \(0.000\), respectively), with no difference in these values between the normal and the CCC groups (\(P = 0.822\) and \(0.667\), respectively).

The predictive value of the variables
We carried out ROC analysis for the sensitivity and the specificity of strain parameters for the presence of CCC. Operational cut-off values with corresponding predictive characteristics are shown in Table 4. At a cut-off value of 0.75, ROC analysis revealed a sensitivity of 75.8% and a specificity of 60.6%, with the area under the curve being 0.747, for the late diastolic strain rate in circumferential direction to predict the presence of CCC (Fig. 4).

Discussion
Few studies have investigated the significant role of 2-D strain detection in evaluating the function of the coronary collateral artery in the ischemic myocardium to predict the presence of the coronary collateral artery. We used 2-D strain to verify the effectiveness of CCC in the ischemic myocardium function and mechanical dysynchrony in patients. Detecting 2-D strain provided a novel and invasive measurement to evaluate coronary collateral function in the ischemic myocardium within the vascular territory supplied by the related artery. The peak late diastolic strain rate in the circumference measured by 2-D strain was the best among all the values to predict the presence of CCC. Using 2-D strain, we detected that

![Fig. 1](https://example.com/fig1.jpg)

The collateral coronary artery from the right coronary arteries to the left anterior descending. The arrow indicates the formation of coronary collateral circulation.
CCC can preserve the systolic and diastolic function of interrelated ischemic myocardium segments and prevent the mechanical dyssynchrony of the heart. Previously reported studies have established that well-grown versus poorly grown collateral arteries in humans may have a beneficial effect on the infarct size.

### Table 2  Strain and time indices of myocardial segments in longitudinal and circumferential directions

<table>
<thead>
<tr>
<th>Variable</th>
<th>Normal</th>
<th>CCC</th>
<th>NCCC</th>
<th>Normal</th>
<th>CCC</th>
<th>NCCC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sps (%)</td>
<td>−17.4 ±3.26</td>
<td>−16.07 ±3.03*</td>
<td>−15.03 ±3.24**</td>
<td>−18.91 ±5.53</td>
<td>−17.77 ±5.20</td>
<td>−15.79 ±5.31**</td>
</tr>
<tr>
<td>Ses (%)</td>
<td>−17.53 ±3.29</td>
<td>−15.78 ±3.02*</td>
<td>−14.70 ±3.22**</td>
<td>−18.05 ±6.67</td>
<td>−17.34 ±5.35</td>
<td>−14.90 ±5.39**</td>
</tr>
<tr>
<td>TSp (%m)</td>
<td>329.92 ±54.88</td>
<td>355.61 ±34.50*</td>
<td>376.20 ±31.92**</td>
<td>325.55 ±54.92</td>
<td>333.10 ±56.99</td>
<td>371.96 ±67.42**</td>
</tr>
<tr>
<td>TSe (%m)</td>
<td>382.28 ±33.79</td>
<td>383.56 ±32.22*</td>
<td>394.55 ±27.36**</td>
<td>358.44 ±43.01</td>
<td>384.76 ±49.44*</td>
<td>411.53 ±68.58**</td>
</tr>
<tr>
<td>TSRe (%m)</td>
<td>555.20 ±48.97</td>
<td>549.59 ±45.61</td>
<td>556.77 ±44.35</td>
<td>516.21 ±55.14</td>
<td>526.72 ±51.98</td>
<td>577.30 ±104.25**</td>
</tr>
<tr>
<td>TSRe (%m)</td>
<td>923.45 ±35.43</td>
<td>916.46 ±38.43</td>
<td>915.97 ±29.85</td>
<td>897.11 ±47.27</td>
<td>904.31 ±54.59</td>
<td>949.73 ±108.05**</td>
</tr>
<tr>
<td>TSRe (%m)</td>
<td>162.47 ±40.03</td>
<td>166.71 ±38.43</td>
<td>167.03 ±46.82**</td>
<td>155.04 ±48.03</td>
<td>178.22 ±61.73*</td>
<td>187.99 ±73.77**</td>
</tr>
</tbody>
</table>

Data are mean ± SD.
CCC, coronary collateral circulation; NCCC, no coronary collateral circulation; Ses, end systolic strain; Sps, systolic peak strain; TSe, time to systolic end strain; TSp, time to the peak systolic strain; TSRe, time to late peak diastolic strain rate; TSe, time to early peak diastolic strain rate; TSRe, time to peak systolic strain rate.

*P<0.05, compared with normal.
**P<0.05, compared with CCC.

### Fig. 2

Strain rate (a, peak systolic strain rate; b, peak early diastolic strain rate; c, peak late diastolic strain rate) in the longitudinal direction. No coronary collateral circulation (NCCC) segments are in the territory of one of the three main arteries, including the right coronary artery, the left anterior descending branch, and the left circumflex branch stenosis; coronary collateral circulation (CCC) segments are in the territory of one of the three main arteries with stenosis. Normal is the control group segments, which are in the territory with artery stenosis less than 20% on coronary artery angiography.
ventricular aneurysm formation, and ventricular function [6–10]. However, previous studies focused on clinical endpoints, and these endpoints are too blunt to discern subtle changes at follow-up. The best technique to evaluate arteries should be accurate and invasively measure the function of the CCC quantitatively.

Table 3  Strain rate and time indices of myocardial segments in radial and rotation directions

<table>
<thead>
<tr>
<th>Variable (ms)</th>
<th>Normal</th>
<th>CCC</th>
<th>NCCC</th>
<th>Normal</th>
<th>CCC</th>
<th>NCCC</th>
</tr>
</thead>
<tbody>
<tr>
<td>SRe (s)</td>
<td>–1.61 ± 0.30</td>
<td>–1.43 ± 0.35*</td>
<td>–1.41 ± 0.45*</td>
<td>52.09 ± 11.73</td>
<td>44.43 ± 23.15*</td>
<td>34.73 ± 14.49* **</td>
</tr>
<tr>
<td>SRa (s)</td>
<td>–1.21 ± 0.32</td>
<td>–1.05 ± 0.38*</td>
<td>–0.98 ± 0.40*</td>
<td>32.67 ± 7.66</td>
<td>26.58 ± 14.80*</td>
<td>25.61 ± 11.54*</td>
</tr>
<tr>
<td>SRs (s)</td>
<td>1.85 ± 0.25</td>
<td>1.77 ± 0.33</td>
<td>1.61 ± 0.44**</td>
<td>46.05 ± 10.55</td>
<td>44.78 ± 15.29</td>
<td>39.40 ± 18.75**</td>
</tr>
<tr>
<td>TSRe (ms)</td>
<td>534.95 ± 60.14</td>
<td>561.86 ± 57.36</td>
<td>601.92 ± 114.53**</td>
<td>495.96 ± 56.33</td>
<td>499.76 ± 72.06</td>
<td>494.04 ± 91.97</td>
</tr>
<tr>
<td>TSRa (ms)</td>
<td>890.72 ± 85.19</td>
<td>907.60 ± 61.37</td>
<td>905.22 ± 120.37**</td>
<td>864.11 ± 78.71</td>
<td>897.44 ± 74.10*</td>
<td>913.70 ± 105.24**</td>
</tr>
<tr>
<td>TSRs (ms)</td>
<td>155.04 ± 48.03</td>
<td>178.22 ± 61.73*</td>
<td>187.99 ± 73.77**</td>
<td>185.06 ± 58.80</td>
<td>187.03 ± 50.41*</td>
<td>251.12 ± 86.40**</td>
</tr>
</tbody>
</table>

Data are mean ± SD.
CCC, coronary collateral circulation; NCCC, no coronary collateral circulation; SRa, late diastolic strain rate; SRe, early diastolic strain rate; SRs, systolic peak strain rate; TSRe, time to late peak diastolic strain rate; TSRa, time to early peak diastolic strain rate; TSRs, time to peak systolic strain rate.
*P<0.05, compared with normal.
**P<0.05, compared with CCC.
2-D strain is a feature-tracking echocardiographic technique that incorporates traditional speckle tracking, tissue–blood border detection, image normalization, and the periodicity of the cardiac cycle using R–R intervals, thus providing more information than qualitative data [11]. The geometric shift of each speckle represents local tissue movement. The motion pattern of myocardial tissue is reflected by the motion pattern of speckles. By tracking these speckles, the strain and the strain rate can be calculated. The technique has been found to be accurate at a minimal temporal resolution of 30 Hz [3,4], which is far below the frame rate used in the current study. Similarly, the accuracy of 2-D strain measurements has been highly reproducible [5].

Using the 2-D strain technique, we demonstrated the association of the peak systolic strain and the strain rate of the ischemic myocardium segments determined by 2-D strain measurement and the presence of CCC as seen on angiography. The ischemic myocardium with CCC had better function than the NCCC myocardium. The peak strain and the systolic strain rate were larger in patients with CCC than those without CCC. Moreover, the normal control and the CCC groups did not differ in some strain values, especially SRe in the longitudinal, circumferential, and radial directions. This finding is consistent with previous reports that among some patients, the CCC is sufficient for maintaining some functions of the ischemic myocardium [6,12,13]. Also, some values could predict the presence of CCC to some extent, especially late diastolic strain rate in the circumferential direction.

Many reports have demonstrated that myocardium dyssynchrony results in decreased cardiac output, slowed relaxation rates, and reduced peak filling velocity, and an increased myocardial energy demand [14–16]. Abnormal patterns of contraction and myocardial stretch likely increase mechanical loading and myocardial work and may impair functional recovery after ischemic injury [17]. Some experimental and clinical results suggest that a ventricular contraction pattern may be an important determinant of prognosis independent of overall ventricular function or regional wall motion abnormalities [18]. Our data showed that CCC helped amend the mechanical asynchrony of the myocardial segment motion. CCC favored myocardial synchronous contraction and in some way improved the left-ventricular function for better prognosis of patients.

CCC plays a considerable role in preserving left-ventricular function. The historical background and the current knowledge of coronary collateral growth in terms of the therapeutic potential is crucial for treating CAD. Long-term and high-grade coronary stenosis is mainly responsible for collateral vessel growth. We found all collaterals present with a stenosis diameter at least 85%, which may be explained by severe coronary narrowing resulting in myocardial ischemia and a pressure gradient between the coronary arteries providing and receiving blood across the collateral network. New establishment of a pressure gradient leads to recruitment of collateral blood flow and increased shear stress at the site of preexisting collateral vessels [6,19,20].

Measuring 2-D strain is an efficient method to assess ischemic myocardial regional function. Also, the values acquired by the technique can predict the presence of CCC. Many elderly patients in the clinic cannot undergo CAG because of bodily or other organ dysfunction, or they may have allergies to contrast agents. Current findings related to nonacute coronary intervention indications have been controversial. Searching for a viable myocardium before revascularization of occluded coronary arteries is recommended, but we lack practical and sensitive methods to assess myocardial viability in the catheterization laboratory. Several prior reports addressed the issue of the

<table>
<thead>
<tr>
<th>Variable</th>
<th>Cut-off point</th>
<th>Sensitivity</th>
<th>Specificity</th>
<th>Area of ROC</th>
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<tbody>
<tr>
<td>LSps</td>
<td>–17.53</td>
<td>0.627</td>
<td>0.536</td>
<td>0.598</td>
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<tr>
<td>LSes</td>
<td>–15.22</td>
<td>0.606</td>
<td>0.531</td>
<td>0.591</td>
</tr>
<tr>
<td>RSes</td>
<td>21.80</td>
<td>0.705</td>
<td>0.518</td>
<td>0.634</td>
</tr>
<tr>
<td>RSRs</td>
<td>1.74</td>
<td>0.621</td>
<td>0.593</td>
<td>0.638</td>
</tr>
<tr>
<td>SRa</td>
<td>0.76</td>
<td>0.742</td>
<td>0.606</td>
<td>0.777</td>
</tr>
<tr>
<td>Rotra</td>
<td>42.74</td>
<td>0.515</td>
<td>0.719</td>
<td>0.641</td>
</tr>
</tbody>
</table>

CSRa, late diastolic strain rate in circumferential direction; LSes, end systolic strain in longitudinal direction; RSps, peak systolic strain in longitudinal direction; ROC, receiver operating characteristic curve; Rotra, late diastolic rotation rate; RSes, end systolic strain in radial direction; RSRs, peak systolic strain rate in radial direction.
presence of collaterals predicting the presence of viability in the supplied region [13,21] and reported that the visualization of collaterals to the infarct-related artery on CAG was an independent predictor of contractile recovery after revascularization. In agreement with our results, measuring 2-D strain may reveal the presence of CCC and may in the future reveal the surviving myocardium to guide coronary reconstruction. 2-D strain was an efficient method to assess ischemic myocardial regional function and was a noninvasive assessment of CCC. Therapeutic augmentation of collaterals with emerging biological therapies can shed light on patients who are not candidates for revascularization procedures.

Conclusion

CCC can significantly improve the systolic and diastolic function of the ischemic myocardium and preserve left-ventricular myocardium dyssynchrony. Measurement of 2-D strain can be efficient in assessing the ischemic myocardial regional function and a noninvasive assessment of CCC. Circumferential SRa might be used to predict the presence of CCC.

Acknowledgements

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Conflicts of interest

There are no conflicts of interest.

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