Myocardial layer-specific analysis of ischemic memory using speckle tracking echocardiography

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Abstract

The assessment of post-systolic shortening (PSS) by speckle tracking echocardiography allows myocardial ischemic memory imaging. Because the endocardial layer is more vulnerable to ischemia, the assessment of this layer might be useful for detecting ischemic memory. Serial echocardiographic data were acquired from 9 dogs with 2 min of coronary occlusion followed by reperfusion. Regional deformation parameters were measured in the risk and normal areas. Using speckle tracking echocardiography, circumferential strain was analyzed in the endocardial, mid-wall, and epicardial layers; and radial strain was analyzed in the inner half, outer half and entire (transmural) layer. In the risk area, peak systolic and end-systolic strain in the circumferential and radial directions significantly decreased during occlusion, but recovered to the baseline levels immediately after reperfusion in all layers. However, circumferential post-systolic strain index (PSI), a parameter of PSS, significantly increased during occlusion, and the significant increases persisted until 20 min after reperfusion in the endocardial and mid-wall layers. Radial PSI tended to increase after reperfusion in the inner half and entire layer but these increases were not significant compared with baseline. In the normal area, systolic strains and PSI in the radial and circumferential directions hardly changed before and after occlusion/reperfusion in all layers. In layer-specific analysis with speckle tracking echocardiography, circumferential PSS in the endocardial and mid-wall layers may be useful for detecting ischemic memory.

Key words: echocardiography, myocardial strain, ischemia, post-systolic shortening

Background

Tissue Doppler and speckle tracking echocardiography are promising methods for the quantitative assessment of regional myocardial deformation. These techniques facilitate the detection of subtle myocardial motion such as post-systolic shortening (PSS), which is difficult to visualize with conventional echocardiography [1, 2]. PSS is defined as myocardial contraction after aortic valve closure (AVC) and is known to be a sensitive marker of myocardial ischemia [3, 4]. Based on experimental dog models with brief myocardial ischemia, we previously reported that PSS persists in the risk area even after rapid recovery of systolic strain abnormalities [5, 6]. These results suggest that the assessment of PSS allows after-the-fact recognition of an ischemic insult and can be used for myocardial ischemic memory imaging.

The left ventricular (LV) wall is composed of endocardial, mid-wall and epicardial layers and deformation is not homogeneous among the layers. Compared with the epicardial layer, the endocardial layer has greater oxygen consumption when coronary perfusion is normal and receives less collateral flow when perfusion is compromised. Therefore, the endocardial layer is more vulnerable to ischemia [7]. Recent studies have shown that speckle tracking echocardiography can assess regional myocardial deformation layer by layer [8-10] and can provide an early diagnosis of myocardial ischemia [11, 12]. However, the value of the layer-specific analysis for assessing myocardial ischemic memory is still unclear.

In this study, we evaluated whether layer-specific analysis with speckle tracking echocardiography is feasible and useful for assessing ischemic memory after brief myocardial ischemia.

Methods

Animal preparation

All animal studies were performed in accordance with guidelines for the care and use of laboratory animals at our institution. A total of 9 open-chest mongrel dogs (14.7 ± 0.6 kg) were used in this study. Dogs were anesthetized with intravenous pentobarbital sodium (35 mg/kg), intubated and ventilated using a respirator pump. Oxygen saturation was monitored by a pulse oximeter and maintained within normal ranges. Anesthesia with pentobarbital sodium was maintained throughout the experiment (5.7-7.7 mg/kg/h). The electrocardiogram was monitored continuously, and LV pressure was measured by a 5-F micromanometer (Millar Instruments, Houston, TX). The heart was suspended in a pericardial cradle through a left lateral thoracotomy in the fifth intercostal space. The proximal portion of the left anterior descending artery (LAD) was dissected free from surrounding tissues, and a vascular occluder was placed around the artery. A perivascular ultrasonic flow probe was placed on the LAD just distal to the occluder and connected to a digital flowmeter (Transonic Systems, Ithaca, NY).

Echocardiography

Echocardiography for the speckle tracking analysis was performed using an Aplio SSA-770A ultrasound system equipped with a PST-50AT transducer (Toshiba, Otawara, Japan). The transmitting and receiving frequency was 5.0 MHz, and the frame rate was 118 frames/s. The LV short-axis view at the papillary muscle level was visualized with a water bath as a standoff. The position of the transducer was fixed by a mechanical arm. Two-dimensional images were digitally captured for each period of data acquisition.

A microphone for the phonocardiogram was placed directly on the base of the aorta. Timing of AVC was determined by the aortic component of the second heart sound derived from the phonocardiogram [2].

Experimental protocol

The LAD was occluded for 2 min, followed by reperfusion for 60 min. Before LAD occlusion, heparin (100 U/kg) was intravenously administrated to prevent coronary thromboembolism. LAD occlusion and reperfusion was confirmed by flow measurement. Echocardiographic data were acquired at baseline, at

the end of occlusion and at 10, 20, 30, and 60 min after the onset of reperfusion. LV pressure was recorded at the same time as the acquisition of the echocardiographic data. LV systolic pressure, LV end-diastolic pressure, and maximum and minimum time derivatives of LV pressure (dP/dt_{max}, dP/dt_{min}) were averaged from 5 consecutive cardiac cycles.

At the end of the protocol, real-time myocardial contrast echocardiography (22 frames/s) was performed during transient LAD occlusion to identify the risk area, as previously described [5]. For contrast microbubbles, Optison (Amersham Health, Princeton, NJ) diluted 1:10 in normal saline was used and administered intravenously at a rate of 60 mL/h.

Data analysis

The 2-dimensional speckle tracking analysis was performed in the clip of captured images by offline software (Toshiba, Otawara, Japan) [10]. End-diastole was defined at peak R-wave of the electrocardiogram and end-systole was defined at the timing of AVC. The endocardial border excluding papillary muscles was manually traced at end-systole. The epicardial border was determined by setting an even width of myocardium. When myocardial thickness was uneven at end-systole, the width was corresponded to that of the risk area. A mid-wall border was automatically determined at the midpoints between the endocardial and epicardial borders at end-diastole.

The LV myocardium was automatically divided into 6 segments and myocardial circumferential and radial strain profiles were analyzed in a segment that located within the risk area identified by myocardial contrast echocardiography and an opposite segment within the normal area (Fig. 1). When the segment was displaced from the risk area, the segment size was adjusted manually. For the layer-specific analysis, in circumferential strain, the length between sampling points on the endocardial, mid-wall, and epicardial tracking borders were measured in this software, respectively. For radial strain, the length between endocardial and mid-wall borders (inner half layer) and the length between mid-wall and epicardial borders (outer half layer) were measured. Radial strain in the transmural layer (entire layer) was also

measured, whereas circumferential strain in the entire layer could not be analyzed due to the algorithm of this software. Zero strain as a reference point was set at end-diastole.

Peak systolic and end-systolic strain values in the circumferential and radial directions were calculated for each layer. When PSS was observed in the profile, post-systolic strain index (PSI) as a parameter of PSS was calculated from the following ratio: (amplitude of PSS) / (maximum amplitude of strain during the cardiac cycle) (Fig. 2). When PSS was not observed, PSI was assigned a value of zero. Peak systolic strain, end-systolic strain and PSI were calculated from 3 consecutive cardiac cycles and averaged. The ability of the deformation parameters in each layer to detect ischemic memory at 10, 20, 30 and 60 min after reperfusion were determined by receiver operating characteristic (ROC) curve analysis.

One image clip was randomly selected from each dog, and a total of 9 clips were used to assess interobserver and intraobserver variability for peak systolic strain and PSI in each layer of the risk area.

Statistical analysis

Data are expressed as mean ± SD. Each hemodynamic and myocardial deformation parameter at the various time points were compared against the values at baseline by one-way analysis of variance followed by a Tukey-Kramer post-hoc test. When the variance was not homogenous, the data were analyzed by the Games-Howell test. These statistical analyses were performed using SPSS (SPSS Inc., Chicago, IL, USA). ROC curve analysis was performed to determine the ability of the strain measurements to detect ischemic memory at each time after reperfusion. The area under the ROC curve (AUC) for peak systolic strain, end-systolic strain and PSI were calculated for each layer of the risk area. These AUCs were compared to the line of no information (AUC = 0.50). The interobserver and intraobserver variability was determined by Bland-Altman analysis. Values of $p < 0.05$ were considered significant.

Results

Because 4 segments in the normal area were excluded from the strain analysis due to insufficient quality of speckle tracking, a total of 9 segments in the risk area and 5 segments in the normal area were included in the final analysis.

Hemodynamics

LV end-diastolic pressure significantly increased, LAD flow significantly decreased and the absolute value of dP/dt_{min} tended to decrease during LAD occlusion compared with their respective baseline values. However, these parameters completely recovered to baseline by 10 min after reperfusion (Table 1).

Serial changes in deformation parameters in layer-specific analysis

Fig. 3 shows changes in the circumferential deformation parameters in the risk and normal areas before and after occlusion/reperfusion. Peak systolic strain at baseline was highest in the endocardial layer, lower in the mid-wall layer and lowest epicardial layer (Fig. 3a and 3b). In the risk area, peak systolic and end-systolic strains significantly decreased during occlusion, but recovered to baseline by 10 min after reperfusion in all layers (Fig. 3a and 3c). PSI significantly increased during occlusion in all 3 layers and the significant increase persisted until 20 min after reperfusion in the endocardial and mid-wall layers, although it did not persist in the epicardial layer (Fig. 3e). In the normal area, systolic strains and PSI hardly changed before and after occlusion/reperfusion in all layers (Fig. 3b, 3d and 3f). Representative circumferential profiles in the risk and normal area derived from layer-specific analysis are shown in Fig. 4.

Fig. 5 shows changes in the radial deformation parameters in the risk and normal areas before and after occlusion/reperfusion. In the risk area, peak systolic and end-systolic strains significantly decreased during occlusion, but recovered to baseline by 10 min after reperfusion in all layers (Fig. 5a and 5c). PSI also significantly increased during occlusion in all 3 layers and tended to increase after reperfusion in the inner half and entire layer, but these increases were not significant compared with baseline because of

large standard deviations (Fig. 5e). In the normal area, systolic strains and PSI hardly changed before and after occlusion/reperfusion in all layers (Fig. 5b, 5d and 5f).

ROC curve analysis of deformation parameters in each layer to detect ischemic memory

For circumferential and radial strains, the AUCs for peak systolic and end-systolic strains in each layer of the risk area were approximately 0.5 at 10, 20, 30 and 60 min after reperfusion. In contrast, the AUC for PSI in each layer tended to be larger than those for peak systolic and end-systolic strains. For circumferential PSI, the AUC in each layer was significantly larger than the line of no information at 10 and 20 min after reperfusion, and the AUCs in the endocardial and mid-wall layers were significant but that in the epicardial layer was not significant at 30 min after reperfusion (Table 2 and Fig. 5). For radial PSI, the AUCs in the inner half and entire layer were significantly larger than the line of no information at 10 min after reperfusion, but only the AUC in the entire layer was significantly larger at 20 min after reperfusion (Table 2).

Interobserver and intraobserver variability

The interobserver and intraobserver variability for peak systolic strain and PSI in each layer of the risk area are shown in Table 3. The limits of agreement for the radial parameters tended to be larger than those for the circumferential parameters.

Discussion

Using a dog model with brief myocardial ischemia, a layer-specific analysis of myocardial strain was performed over time in this study. Peak systolic and end-systolic strain did not demonstrate ischemic memory in any layer. In contrast, circumferential PSI in the endocardial and mid-wall layers were better parameters for assessing ischemic memory than that in the epicardial layer. Because of the high variability (SD) of radial strain in the inner half of the LV wall, radial PSI in the inner half was not suitable for assessing ischemic memory.

Assessment of myocardial ischemic memory using echocardiography

Chest pain often resolves before a patient presents to the hospital, which makes it difficult to accurately determine whether the chest pain was due to myocardial ischemia. Myocardial ischemic memory imaging, denoting the visualization of abnormalities provoked by ischemia and sustained even after restoration of perfusion, is therefore desirable. Although cardiac single-photon emission computed tomography with beta-methyl-p-(123I)-iodophenyl-pentadecanoic acid (BMIPP) is thought to be a promising method for ischemic memory imaging [13-15], there is a limitation that it must be performed in a radiation-controlled area. If ischemic memory can be assessed by echocardiography, it would have greater utility because it can be done at the bedside. In previous studies with tissue Doppler [5] and speckle tracking echocardiography [6], we reported that PSS persists after recovery from brief myocardial ischemia in a dog model. These findings suggest that ischemic memory imaging is feasible with echocardiography.

Because the endocardial layer is more vulnerable to ischemia, a layer-specific analysis should be important to detect myocardial ischemia. Several studies demonstrated that layer-specific analysis allows the accurate detection of ischemic myocardium using tissue Doppler [16, 17] and speckle tracking echocardiography [11, 12]. However, the usefulness of layer-specific analysis for assessing ischemic memory has never been evaluated.

Layer-specific analysis of ischemic memory

Peak systolic strain and end-systolic strain

In the stunned myocardium, systolic dysfunction remains despite normalization of myocardial blood flow. The assessment of such dysfunction can be used as a measure of ischemic memory. Bolli et al. showed that the recovery of systolic dysfunction after 15 min of coronary occlusion is delayed in the endocardial layer compared with the epicardial layer [18]. A layer-specific analysis of systolic dysfunction might have an advantage to detect ischemic memory for this reason. However, in the present study, abnormalities of peak systolic and end-systolic strain in the circumferential and radial directions did not persist after

reperfusion in any layer after 2 min of occlusion. Even if layer-specific analysis is performed, it seems to be difficult to detect ischemic memory from systolic dysfunction alone when the period of myocardial ischemia is very brief (2 min).

PSS

As mentioned above, PSS can indicate ischemic memory after brief myocardial ischemia. In a previous study using speckle tracking echocardiography, circumferential PSS persisted longer than radial PSS, although PSS could be observed in both the circumferential and radial directions [6]. Since circumferential strain reflected only endocardial deformation and radial strain reflected transmural deformation in the previous software, we predicted that the analysis of PSS in the endocardial layer might be advantageous for assessing ischemic memory.

Wang et al. evaluated regional myocardial work from pressure-strain loops in dogs with progressive coronary stenosis. In that experiment, PSS-related work was significantly changed in the endocardial layer with a decrease in endocardial myocardial blood flow despite no significant change in the entire layer of ischemic myocardium [19]. Their data seem to demonstrate an advantage of PSS assessment in the endocardial layer.

In the present study, the increase in circumferential PSI after reperfusion persisted longer in the endocardial and mid-wall layers than in the epicardial layer. ROC curve analysis for detecting ischemic memory showed that the AUCs for circumferential PSI in the endocardial and mid-wall layers were larger than the AUC in the epicardial layer. These results suggest that layer-specific analysis might increase the diagnostic accuracy of PSI to detect ischemic memory. On the other hand, radial PSI tended to increase after reperfusion in the inner half and entire layer, but this increase was not significant. The AUC for radial PSI in the inner half was similar to the AUC for the entire layer. The failure of radial PSI to detect ischemic memory may be due to the larger standard deviations of radial PSI, as shown in Fig. 5. Radial PSI is also prone to have larger limits of agreement in variability than circumferential strain [6], because

of the small number of sampling points in the radial direction. We therefore recommend the assessment of circumferential PSI in the endocardial layer for detecting ischemic memory.

Although radial PSI in the entire layer significantly increased 10 min after reperfusion in our previous study [6], it did not significantly increase in the present study. This discrepancy could be explained by difference between the perfusion territory of the left circumflex artery (LCx) and that of LAD. Because the LAD perfusion territory is smaller than the LCx territory in dogs, the risk area in the present study (LAD) was apparently smaller than that in our previous study (LCx). The size of the risk area may influence the duration of ischemic memory, as assessed by the persistence of PSS.

The mechanisms underlying PSS remain unclear. Claus et al. have demonstrated that the passive interaction between myocardial segments can explain the occurrence of PSS [20]. However, the persistence of PSS in our study could not be explained by only passive interaction because peak systolic and end-systolic strains in the risk area recover beyond that in the normal area after reperfusion. Lucats et al. have suggested that PSS is associated with pre-stretch, which is observed during the early systolic phase in the myocardium with delayed or impaired contraction [21]. We think that the influence of pre-stretch should be examined in further studies.

Study limitations

In this study, we assessed circumferential and radial but not longitudinal strains. This reason is due to the less gradient of longitudinal strain among the layers [9].

We excluded 4 segments in the normal area from the strain analysis due to insufficient quality of speckle tracking. There were few speckles in the excluded segments because ultrasound beams tended to be parallel to the main myocardial fiber orientation in these segments. The presence of speckles within the myocardium seems to be necessary for accurate tracking, especially in the mid-wall layer.

In this study, circumferential strain was calculated from the sampling points on only one tracking line. Consequently, circumferential strain in the entire layer could not be analyzed. Thus, it is unknown

whether endocardial circumferential strain is superior to circumferential strain in the entire layer for assessing ischemic memory. Because there is variability in speckle tracking measurements among echocardiographic systems [22], evaluation of PSI by other systems should be performed in the future.

Conclusions

In layer-specific analysis with speckle tracking echocardiography, PSS derived from circumferential strain persisted longer after brief myocardial ischemia in the endocardial and mid-wall layers than in the epicardial layer. The endocardial layer had higher accuracy for detecting the persistence of PSS. Layer-specific analysis is feasible and useful for assessing ischemic memory.

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Figure legends

Figure 1: Layer-specific analysis with speckle tracking echocardiography

(a) The risk area was identified by real-time myocardial contrast echocardiography during transient LAD occlusion. With speckle tracking echocardiography, the myocardium in the short axis was automatically divided into 6 segments, and strain profiles were analyzed in the risk and opposite normal areas. (b) Circumferential strain was measured in endocardial, mid-wall, and epicardial layers. (c) Radial strain was measured in inner half, outer half, and entire (transmural) layer.

Figure 2: Parameters derived from circumferential strain profile

Peak systolic strain (ε_s) and end-systolic strain (ε_{es}) were measured. Post-systolic strain index (PSI) was calculated from the following ratio: amplitude of PSS (ε_{pss})/ maximum amplitude of strain during the cardiac cycle (ε_{max}). End-systole is indicated by the red dotted line.

Figure 3: Changes in the circumferential deformation parameters before and after occlusion/reperfusion

In the risk area, peak systolic and end-systolic strains decreased during occlusion but recovered to baseline by 10 min after reperfusion in all 3 layers (a, c). Post-systolic strain index (PSI) significantly increased during occlusion, and the significant increase persisted until 20 min after reperfusion in the endocardial and mid-wall layers (e). In the normal area, systolic strains and PSI hardly changed before and after occlusion/reperfusion in all layers (b, d, f). *$p < 0.05$ versus baseline.

Figure 4: Circumferential strain profiles derived from layer-specific analysis

In the risk area (blue lines), peak systolic strain (blue circles) decreased and PSS (red arrows) occurred during occlusion in all 3 layers. After reperfusion, peak systolic strain immediately recovered to baseline

in all 3 layers. In contrast, PSS remained until 30 min after reperfusion in the endocardial and mid-wall layers. In the epicardial layer, PSS persisted until 10 min after reperfusion, but could not be detected thereafter. In the normal area (orange lines), peak systolic strain and PSS did not change before and after occlusion/reperfusion in all layers.

Figure 5: Changes in the radial deformation parameters before and after occlusion/reperfusion

In the risk area, peak systolic and end-systolic strains decreased during occlusion but recovered to baseline by 10 min after reperfusion in all 3 layers (a, c). PSI also significantly increased during occlusion in all 3 layers and tended to increase after reperfusion in the inner half and entire layer, but these increases were not significant compared with baseline (e). In the normal area, systolic strains and PSI hardly changed before and after occlusion/reperfusion in all layers (b, d, f). *$p < 0.05$ versus baseline.

Figure 6: Receiver operating characteristic curve analysis of circumferential post-systolic index

The AUC in each layer was significantly larger than the line of no information at 10 and 20 min after reperfusion, and the AUCs in the endocardial and mid-wall layers were significant, but that in the epicardial layer was not significant at 30 min after reperfusion. *$p < 0.05$ versus line of no information (AUC = 0.5).