Usefulness of Myocardial Parametric Imaging to Evaluate Myocardial Viability in Experimental and in Clinical Studies.

Running title: Parametric imaging to evaluate viability

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Key words: parametric imaging, real-time contrast echocardiography, myocardial perfusion
Abstract

Background: The current clinical assessment of myocardial viability by myocardial contrast echocardiography (MCE) is based either on visual interpretation, which is subjective, or on quantitative off-line processing, which is time consuming and lacks spatial resolution. The purpose of this study was to evaluate whether myocardial parametric imaging (MPI) is superior to visual assessment for the evaluation of myocardial viability.

Methods and Results: MCE was assessed in 11 pigs before, during and after left anterior descending coronary artery occlusion and in 32 patients with ischemic heart disease using intravenous SonoVue® administration. In experimental studies we sought to compare 1) perfusion defect area assessment by MPI with 2) visually guided perfusion defect planimetry. Histological assessment of necrotic tissue deemed as the standard reference. In clinical studies viability was assessed on a segmental level by 1) visual analysis of myocardial opacification, 2) quantitative estimation of myocardial blood flow using regions of interest and 3) by MPI. Functional recovery between 3 and 6 months after revascularization deemed as standard reference. In experimental studies, planimetric assessment of infarct size by MPI correlated significantly closer with histology (r²=0.92 versus r²=0.56) and showed lower intraobserver variability (4% versus 15%, p<0.05) compared with visually guided perfusion defect planimetry. In clinical studies, MPI showed higher specificity (66% versus 43%, p<0.05) than visual MCE and good accuracy (81%) for viability detection. It was less time consuming (3.4±1.6 versus 9.2±2.4 minutes per image, p<0.05) than quantitative blood flow estimation by regions of interest and increased the agreement between observers interpreting myocardial perfusion (κ=0.87 versus κ=0.75, p<0.05).

Conclusion: MPI is useful for the evaluation of myocardial viability both in animals and in patients and is less time consuming than quantification analysis by regions of interest and less observer dependent than visual analysis. Thus, strategies incorporating this technique may be valuable for the evaluation of myocardial viability in the clinical routine.
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Introduction

Myocardial contrast echocardiography (MCE) has provided new insight into the assessment of myocardial viability in acute ischemic syndromes (1, 2) and in stable ischemic heart disease (3, 4). However, the current clinical assessment of myocardial viability by MCE is based on visual interpretation of MCE images or on quantitative computer assisted processing. Visual interpretation of myocardial perfusion is subjective and requires experienced investigators (3-7). Quantitative analysis of myocardial perfusion using replenishment kinetics to fit exponential curves is, on the other hand, time consuming and provides low spatial resolution. Myocardial parametric imaging (MPI) is a new method which provides automated color-encoded quantification of myocardial blood flow and parametric imaging according to the relative degree of perfusion (8-9). The feasibility of myocardial parametric quantification to detect inducible ischemia has been currently demonstrated (8, 9). However, there is still no published report on the value of MPI for myocardial viability assessment. The purpose of this study was to assess the value of MPI for viability detection in the experimental setting of acute infarction and in patients with stable ischemic heart disease.

Methods

Experimental studies

Experimental protocol. The experimental protocol was approved by the German Animal Research Committee and carried out as described previously (1, 2). Briefly, 11 farm pigs (15-25 kg) were anaesthetized with alpha chloralose (25 mg/kg/h). The heart was exposed through a sternotomy and an occluder was placed between the first and second diagonal branch of the LAD for 45 minutes followed by 2 hours of reperfusion.

Myocardial contrast echocardiography. Real-time MCE was performed with an ATL HDI 5000 (Philips Medical system, Bothell, WA) in short-axis sections at baseline, during
occlusion and after 2 hours of reperfusion (2). Ultrasound images were performed in color-coded harmonic power pulse inversion (PPI) mode. The mechanical index (MI) was set between 0.14 and 0.18 and the dynamic range was set at “low”. SonoVue® (Bracco, Byk-Gulden, Konstanz, Germany) was applied as continuous intravenous infusion (60ml/h). When the myocardium of the left ventricle was fully opacified a brief pulse of higher mechanical index (flash) was transmitted to “clear” the myocardium from microbubbles. Returning immediately to low-power imaging, replenishment was visualized over 10 to 15 cardiac cycles. The risk area was identified as the area of severely reduced or absent myocardial opacification during LAD occlusion and determined by visually- and by MPI-guided planimetry. Similarly, perfusion defects at 2 hours of reperfusion were determined by planimetry as the area of severely reduced or absent myocardial opacification, 10-15 cardiac cycles after flash and expressed as percentage of the risk area (perfusion-defect-size-to-risk-area-ratio).

**Histology.** The LAD was reoccluded at the end of the experiment and Monastral blue dye (Sigma, St. Louis, MO) was injected into the left atrium to outline the area at risk as previously described (2). Myocardial tissue was then immersed in a solution of 1.3% 2,3,5-triphenyltetrazolium chloride (TTC, Sigma) for 20 min, staining viable myocardium red. The risk area by histology was expressed as percentage of the total left ventricle and the infarct size was expressed as percentage of the risk area and was correlated with the perfusion-defect-size-to-risk-area-ratio estimated by MCE using linear regression analysis and Bland-Altman statistics.

**Clinical studies**

**Patient population.** The study population consisted of 34 consecutive patients with impaired left ventricular function (ejection fraction ≤40%) due to ischemic heart disease, who were scheduled for revascularization. Two patients were excluded due to extremely poor echographic windows. The study was approved by the local ethics committee and all patients
signed informed consent before their enrolment in the study. MCE studies were performed within 2 weeks before revascularization.

**Two-dimensional echocardiography.** Imaging was performed in standard apical views using an ATL HDI 5000. Regional function was assessed according to the 16-segment left ventricular model of the American Society of Echocardiography and graded from 1 (normal), 2 (hypokinesia), 3 (akinesia) to 4 (dyskinesia). Ejection fraction was quantified using the biplane Simpson’s method (10). Follow-up echocardiograms were obtained at 3 to 6 months of follow-up and recovery of regional function was defined as an improvement of ≥1 grade in wall motion. Functional recovery deemed as the standard reference for myocardial viability (3-4, 11).

**Real-time Myocardial Contrast Echocardiography.** Perfusion imaging was performed similarly to experimental studies using low power imaging (4-7). Slow bolus injections of SonoVue® (1.0 to 1.5 ml per bolus) were used to obtain optimal visualization of the left ventricle.

**Visual analysis.** Images were analyzed by 2 independent observers. Afterwards, a third independent observer proved the variability of the 2 first observers and resolved differences in opinion by consensus of all 3. Normally, it takes about 5 to 6 cycles at rest to completely replenish the ultrasound beam in systole after microbubble destruction (4, 7). Therefore, for visual assessment of myocardial viability, readers interpreted interflash sequences concentrating on myocardial opacification in the first 5-6 end-systolic frames after Flash. Segmental opacification was graded using a semi-quantitative scale, as previously described (4-7): 3=homogenous, 2=mildly reduced, 1=severely reduced or absent. Segments with homogenous or mildly reduced opacification were judged as viable, whereas regions with severely reduced or absent opacification were judged as nonviable (4). In order to prevent the effects of attenuation and lateral dropout from affecting interpretation, contrast defects were considered present only when the segment under question was entirely visualized from epicardium to endocardium. A
slight oblique orientation of the four- and two chamber view was obtained when obvious attenuation artefacts in the lateral- or anterior basal segments were seen. If a contrast defect was limited to the anterior- or lateral basal segment the defect was attributed to attenuation.

**Quantitative analysis using regions of interest.** For quantitative analysis regions of interest were placed from epi- to endocardium in 16 myocardial segments. Contrast intensity after flash was measured end-systolic (12). Plots of contrast intensity versus time were constructed and fit to an exponential function \( y=A\left(1-e^{-\beta t}\right) \) (13). The plateau of signal intensity (A) and the slope of maximal signal intensity rise (\( \beta \)) were measured and the product of \( A\beta \) was calculated. Receiver operating characteristics analysis of flow parameters was performed to select the best cut-off value for viability detection.

**Parametric imaging.** Parametric quantification was assessed off-line using commercially available software (Q-Lab, Philips Medical system, Bothell, WA). To generate parametric images, end-systolic frames after FLASH were automatically selected by ECG and an assisted border detection algorithm was employed to define the endo- and epicardial borders. The exponential curve function \([y=A(1-e^{-\beta t})]\) was fitted to the intensity vector from each pixel position and A- and \( \beta \)-values were automatically tabulated. The mean parameters within the myocardial border were calculated and the parameter values from each pixel were colour-encoded according to the mean values. Parametric images of blood volume (A-images), velocity (\( \beta \)-images) and flow (A*\( \beta \)-images) were generated for each pixel in the myocardium. A median filter was used to reduce noise caused by poor curve fits to noisy intensity vectors. Gradations of colour were applied linearly with fixed breakpoints at the mean (green), two-thirds of the mean (yellow) and one-third of the mean (red) corresponding to normal, mildly reduced, severely reduced and absent myocardial opacification by visual criteria.
Methodological approach to compare parametric imaging with standard methods for the assessment of myocardial viability. 

a) In experimental studies. Regions with the lowest myocardial blood flow (≤one-third of the mean) are coded as red or dark red by MPI. We hypothesized that these segments correspond to the area at risk during coronary occlusion and to tissue necrosis at 2 hours after reperfusion. By this approach we sought to compare MPI-guided with visually-guided planimetry for perfusion defect area assessment. Histological analysis of necrotic tissue deemed as the standard reference.

b) In clinical studies. In clinical studies myocardial viability by MPI was assessed qualitatively on a segmental level. We hypothesized that segments coded red or dark red would have low probability to recover contractile function after revascularization (nonviable segments). Thus, segments predominantly coded red or dark red were classified as nonviable and segments predominantly coded yellow or green were identified as viable. By this approach we sought to compare the accuracy of visual analysis with quantitative estimation of blood flow by regions of interest and with MPI. Functional recovery at 3-6 months of follow-up deemed as the standard reference. We also assumed that by this approach the reproducibility and the accuracy of MCE to detect viable myocardium could be increased. The time spent for the assessment of myocardial perfusion by each of the three methods was measured in 20 representative images.

Statistical Analysis. Data are presented as mean ± standard deviation. Agreement between observers was assessed using kappa statistics (14). Visually guided planimetry of perfusion defect area was compared with MPI and with standard techniques using linear regression analysis and Bland-Altman statistics (15). Receiver operating characteristics were used to assess the diagnostic value of quantitative MCE parameters and differences in diagnostic value were evaluated by McNemar χ² test (4-7). Differences were considered significant at p<0.05.
Results

Experimental studies

**Determination of perfusion-defect-size-to-risk-area-ratio.** MPI allowed objective risk area delineation during LAD occlusion and infarct size determination during reperfusion (Fig. 1). MPI of myocardial blood flow (A*β-images) correlated significantly closer with histological perfusion-defect-size-to-risk-area-ratio ($r^2=0.92$, $p<0.0001$) compared with MPI of A-images ($r^2=0.66$, $p<0.01$), with MPI of β-images ($r^2=0.63$, $p<0.01$) and with visually guided planimetry ($r^2=0.56$, $p<0.01$), (Fig. 2).

**Inter- and intraobserver variability.** Determination of the perfusion-defect-size-to-risk-area-ratio by MPI demonstrated lower inter- (6% vs. 18%, $p<0.05$) and intra-observer (4% vs. 15%, $p<0.05$) variabilities compared with visually guided planimetry.

Clinical studies

**Viability detection by parametric imaging in humans.** Parametric quantification of myocardial blood volume (A-images) yielded significantly higher specificity (66% versus 43%, $p<0.05$) compared with visual assessment. Clinical characteristics of patients undergoing revascularization are illustrated in Table 1. Visual analysis was feasible in 457 (89%) of 512 available segments and quantification analysis by time intensity curves and by MPI was feasible in 421 (82%) segments. Selecting cut-off values of $A=0.95$dB for myocardial blood volume and $A^*β=0.28$dB/s for blood flow, failed to significantly improve the diagnostic characteristics of visual assessment (Table 2 and 3 and Fig. 3). However, parametric quantification of blood volume (A-images) yielded significantly higher specificity (66% vs. 43%, $p<0.05$) for viability detection compared with visual assessment and provided good accuracy of 81% (Table 2). Of segments judged as viable according to predefined criteria by MPI, 179 were coded green...
(normal perfusion) and 119 were coded yellow (patchy perfused). Of 179 segments coded green, 150 (84%) showed functional recovery, while of 119 segments showing patchy perfusion by MPI 102 (86%) showed recovery of resting wall motion at follow-up (Table 3). MPI of myocardial blood flow (A*β-images) underestimated myocardial viability resulting in lower sensitivity (72%). Figure 4 shows a patient with an akinetic apex 6 months after revascularization. MCE before revascularization detected the nonviable tissue of the apex showing severely reduced opacification. MPI of myocardial blood volume (A-image) accurately and objectively identified nonviable tissue in the same region, coded as red. MPI of velocity and flow (β and A*β-images) underestimated myocardial viability in the lateral wall.

**Inter- and intraobserver variabilities and time-spent.** Agreement was significantly higher between observers interpreting MCE images by MPI compared with visual analysis (95% (κ = 0.87) versus 82% (κ = 0.75), p<0.05). MPI resulted in similar time spent compared with visual analysis (3.4±1.6 versus 2.7±1.7 minutes per image, p=NS), but in significantly less time spent compared with quantitative analysis, using regions of interest (3.4±1.6 versus 9.2±2.4 minutes per image, p<0.05).

**Discussion**

Myocardial parametric imaging is less time-consuming than off-line processing by regions of interest, less operator dependent than visual analysis and shows high spatial resolution. MPI correlates closer with standard techniques for viability assessment in experimental studies and demonstrates more preferable specificity for viability detection in humans.

**Experimental studies.** Linka et al (8) first introduced parametric analysis of contrast images using peak intensity and peak rate of rise of intensity. This approach was based upon the use of triggered imaging modalities and requires background subtraction of individual frames obtained during increasing trigger intervals to select contrast specific signals. High power
imaging modalities require end-systolic triggering over several seconds. Stable imaging conditions to prevent patient and observer related motion artefacts are therefore mandatory (16). Such artefacts can be more easily recognised and avoided with newer real-time techniques. Furthermore, as tissue nonlinearity can be ignored in low power imaging (17), baseline subtraction is deemed unnecessary by real-time MCE. We have previously demonstrated the potential of real-time MCE to assess the dynamics of postischemic microvascular dysfunction (2, 18) and the usefulness of first-harmonic Fourier algorithms to display myocardial perfusion in a parametric format, which allows an objective evaluation of real-time MCE in animal studies (19).

In the present study we used a simple, commercially available software tool to quantify tissue viability in the experimental setting of myocardial infarction. Visual MCE confirmed ischemia during LAD occlusion and reappearance of a marked perfusion defect 2 hours after reperfusion. Visual assessment of risk area and infarct size corresponded with standard histological techniques. We have previously reported an $r^2$ value of 0.86 for visually guided planimetry in pigs receiving soluble P-selectin to reduce myocardial ischemia-reperfusion injury (2). In the present study no cardioprotective agents were applied so that perfusion defect size was larger. This may have influenced the variability in visually guided perfusion defect planimetry, resulting in a lower $r^2$ value of 0.56. Nevertheless, visually guided planimetry was still significantly related with histological findings ($p<0.01$). As the variability for perfusion defect assessment by MPI was significantly lower, the correlation coefficient between MPI and histology remained high ($r^2=0.92$). Thus, perfusion assessment by MPI demonstrated higher reproducibility, was less time consuming and was closer related to standard histological measurements. Reperfusion of infarcted tissue results in acute microvascular obstruction and vasoconstriction causing decrease in both blood velocity and volume (2). Thus, parametric quantification of $A*\beta$-images incorporating information of both blood volume and velocity offered better correlation of
perfusion defect size assessment than parametric imaging of blood volume or velocity alone (A or β-images). Reperfusion injury occurs in a substantial number of patients with acute coronary syndromes despite the application of aggressive reperfusion treatments. Consequences are impaired reperfusion at the microcirculatory level, contractile dysfunction and poorer clinical outcomes (20). New pharmacological approaches aim at preservation of endothelial function and at inhibition of leukocyte mediated inflammation and vasodilatation (1-2). Since real-time MCE and particularly parametric quantification can assess the patterns of myocardial reperfusion, MPI may become a valuable and practical tool to assess the effectiveness of new pharmacologic or mechanic approaches in acute ischemic syndromes.

**Clinical studies.** In patients with ischemic heart disease revascularization is associated with improvement of left ventricular function and better clinical outcomes, when viability is present (21). We and others previously evaluated the role of MCE in detecting myocardial viability in stable ischemic heart disease using intravenous contrast administration (3-4). However, there is still to the best of our knowledge no published report investigating the value of MPI for viability assessment in humans. Having demonstrated the usefulness of MPI to assess myocardial tissue viability in experimental studies we sought to determine the potential of parametric quantification to improve the diagnostic accuracy of visual MCE for viability detection in patients with ischemic heart disease. Assessment of tissue perfusion by both triggered and real-time techniques suffers from several restrictions, including inhomogeneous distribution of regional contrast opacification, attenuation, contrast drop out in basal segments and blooming (24). These limitations reduce the specificity of MCE to detect myocardial viability by visual criteria (3-4, 22-23). We and others have previously shown that complex and time consuming quantification of regional myocardial blood flow parameters including normalization of regional myocardial blood volume to normal myocardium are necessary, in order to improve
the specificity of the technique (3-4). Chronic ischemic heart disease is associated with myocardial fibrosis and increased collagen content causing decrease in capillary density and particularly of blood volume (3). Thus, it is not surprising that parametric quantification of blood volume provided the most preferable specificity for viability detection in accord with previous clinical studies (4). Myocardial parametric quantification provides automatic calculation of contrast replenishment kinetics in multiple pixels of the myocardium, which are then colour-encoded on a linear scale according to a normal value adjusted to the entire myocardium. This is the first study to demonstrate that by parametric quantification of blood volume heterogeneity of contrast distribution can be reduced and the specificity of the technique for viability detection can be significantly improved from 43\% to 66\%. However, the sensitivity of MPI to detect viability was comparable to visual assessment (88\% versus 86\%) and the increase in accuracy was not statistically significant by McNemar $\chi^2$-test (81\% versus 73\%, $p=\text{ns}$). As parametric imaging demonstrates lower observer variability and requires less experience for the interpretation of myocardial perfusion this method may be of particular interest for novice interpreters.

\textbf{Limitations.} In accord with previous studies, we chose recovery of contractile function after revascularization as a standard reference for assessment of myocardial viability (4, 11) because the latter is an important clinical goal. However, it should be recognized that microvascular integrity in the mid- and epicardial layer may be clinically important in other ways such as protection against left ventricular remodeling even in absence of functional recovery (25). Furthermore, we used slow bolus injections for contrast agent administration in clinical studies, where delivery of microbubbles is not constant and may confound calculations of the peak rate of rise of contrast intensity. The use of continuous infusion may have provided more accurate results for estimation of myocardial blood flow. However, we and others have shown that calculation of replenishment kinetics is still feasible by slow bolus injections during optimal myocardial
opacification (4-6, 26), which may therefore represent a more practical approach in routine clinical settings. Parametric imaging demonstrated a relative high false negative rate, resulting in a moderate negative predictive value of 73% for the viability assessment by myocardial blood volume. However, this was not lower than the negative predictive value achieved by visual assessment in the present and in previous studies using the same technique (4). Due to the relative low specificity and moderate NPV of the technique for prediction of recovery of resting wall motion we have previously suggested that MCE should be combined with low-dose dobutamine stress echocardiography for detection of myocardial viability in order to guide clinical decision making in such patients (4).

**Conclusion.** Myocardial parametric imaging shows lower observer variability compared with visual assessment and is less time-consuming compared with myocardial blood flow quantification using regions of interest. It correlates stronger with standard techniques for the assessment of myocardial viability in experimental studies and demonstrates more preferable specificity for viability detection in humans compared with visual analysis. Thus, methods that incorporate myocardial parametric quantification may be useful for the evaluation of myocardial viability both in animals and in patients and can be utilized in a busy clinical laboratory with the use of commercially available software and contrast agents.
Figure Legends

Fig. 1: Parametric quantification of myocardial blood flow (A*β) allowed objective delineation of the risk area during LAD occlusion and determination of dynamics of infarct size during reperfusion.

Fig. 2: Parametric quantification of myocardial blood flow (A*β) correlated significantly closer with histological assessment of perfusion-defect-size-to-risk-area-ratio (b) compared with visually guided planimetry of perfusion defects (a). Parametric quantification showed lower deviation from the standard techniques compared with visually guided planimetry, as demonstrated by Bland-Altman plots (c and d).

Fig. 3: Receiver characteristic analysis of myocardial blood volume (AUC=0.72) and flow (AUC=0.69) by regions of interest provided good sensitivities (86% and 82%) but low specificities (56% and 53%) for detection of myocardial viability.

Fig. 4: MCE before revascularization accurately detected nonviable tissue in the apical regions, in a patient with an akinetic apex 6 months after revascularization (top, left). MPI of myocardial blood volume (A-Image) also correctly and objectively delineated nonviable myocardium in the apical regions, coded as red (top, right). However, MPI of myocardial blood velocity and flow (β and A*β-Images) underestimated the presence of myocardial viability in the lateral wall (bottom).


Table 1. Demographic and clinical characteristics

<table>
<thead>
<tr>
<th>Patient characteristic</th>
<th>No of patients (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>n = 32</td>
<td></td>
</tr>
<tr>
<td>Age (mean ± standard deviation)</td>
<td>65 ± 8 yrs</td>
</tr>
<tr>
<td>Male sex</td>
<td>30 (94%)</td>
</tr>
<tr>
<td>Single vessel disease</td>
<td>3 (9%)</td>
</tr>
<tr>
<td>Multi vessel disease</td>
<td>29 (91%)</td>
</tr>
<tr>
<td>Coronary artery bypass graft surgery</td>
<td>27 (84%)</td>
</tr>
<tr>
<td>Percutaneous coronary intervention</td>
<td>5 (16%)</td>
</tr>
<tr>
<td>Ejection fraction before revascularization</td>
<td>31 ± 6%</td>
</tr>
<tr>
<td>Ejection fraction after revascularization</td>
<td>39 ± 8%</td>
</tr>
<tr>
<td>Improvement of heart failure symptoms after 6 months</td>
<td>23 (72%)</td>
</tr>
<tr>
<td>Hypertension</td>
<td>29 (91%)</td>
</tr>
<tr>
<td>Diabetes</td>
<td>14 (44%)</td>
</tr>
<tr>
<td>Elevated cholesterol level (LDL&gt;120 mg/dl)</td>
<td>29 (91%)</td>
</tr>
<tr>
<td>Family history of CAD</td>
<td>17 (53%)</td>
</tr>
<tr>
<td>Tobacco use</td>
<td>21 (66%)</td>
</tr>
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</table>
Table 2. Prediction of myocardial viability by visual, quantitative contrast echocardiography and by parametric imaging

<table>
<thead>
<tr>
<th>Method</th>
<th>Sensitivity</th>
<th>Specificity</th>
<th>PPV</th>
<th>NPV</th>
<th>Accuracy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Visual assessment</td>
<td>86%</td>
<td>43%</td>
<td>77%</td>
<td>57%</td>
<td>73%</td>
</tr>
<tr>
<td>Quantitative A-values (cut-off=0.95 dB)</td>
<td>86%</td>
<td>56%</td>
<td>80%</td>
<td>65%</td>
<td>76%</td>
</tr>
<tr>
<td>Quantitative A*β-values (cut-off=0.28 dB/s)</td>
<td>82%</td>
<td>53%</td>
<td>79%</td>
<td>59%</td>
<td>73%</td>
</tr>
<tr>
<td>Parametric quantification, A-images</td>
<td>88%</td>
<td>66%*</td>
<td>85%</td>
<td>73%</td>
<td>81%</td>
</tr>
<tr>
<td>Parametric quantification, A*β-images</td>
<td>72%#</td>
<td>62%</td>
<td>73%</td>
<td>61%</td>
<td>68%</td>
</tr>
</tbody>
</table>

*, p < 0.05 visual assessment versus MPI of A-Images, #, p < 0.05 MPI of A versus A*β-Images

PPV indicates positive predictive value and NPV negative predictive value
Table 3. Contingency tables comparing visual, quantitative MCE and MPI for detection of functional recovery

<table>
<thead>
<tr>
<th>Wall motion (FU)</th>
<th>Viable</th>
<th>Non-viable</th>
<th>Viable</th>
<th>Non-viable</th>
<th>Viable</th>
<th>Non-viable</th>
</tr>
</thead>
<tbody>
<tr>
<td>Recovery</td>
<td>272</td>
<td>46</td>
<td>245</td>
<td>41</td>
<td>234</td>
<td>51</td>
</tr>
<tr>
<td>Non-recovery</td>
<td>79</td>
<td>60</td>
<td>60</td>
<td>75</td>
<td>64</td>
<td>72</td>
</tr>
</tbody>
</table>

κ Agreement

Visual A-values | A*β-values

<table>
<thead>
<tr>
<th>Wall motion (FU)</th>
<th>Viable</th>
<th>Green</th>
<th>Yellow</th>
<th>Non-viable</th>
<th>Viable</th>
<th>Non-viable</th>
</tr>
</thead>
<tbody>
<tr>
<td>Recovery</td>
<td>252</td>
<td>150</td>
<td>102</td>
<td>33</td>
<td>179</td>
<td>69</td>
</tr>
<tr>
<td>Non-recovery</td>
<td>46</td>
<td>29</td>
<td>17</td>
<td>90</td>
<td>65</td>
<td>108</td>
</tr>
</tbody>
</table>

κ Agreement

FU indicates follow-up
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