An assessment of dobutamine echocardiography and end-diastolic wall thickness for predicting post-revascularization functional recovery in patients with chronic coronary artery disease

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Aims This study investigated the functional outcome of wall-thinned, akinetic myocardium after revascularization and evaluated the accuracy of dobutamine echocardiography in predicting post-revascularization functional recovery with the assessment of end-diastolic wall thickness in chronic ischemic patients.

Methods and Results: Fifty-three patients underwent dobutamine echocardiography before coronary revascularization. End-diastolic wall thickness was also evaluated before and after revascularization. The sensitivity and specificity of dobutamine echocardiography to predict post-revascularization functional recovery were 69% and 100% in 58 akinetic/dyskinetic segments, and 86% and 57% in 96 hypokinetic segments. Of 19 akinetic/dyskinetic segments with a preserved end-diastolic wall thickness, 17 (89%) showed functional recovery after revascularization, and dobutamine detected recovery in 14 (83%) of these 17 segments. Of 39 akinetic/dyskinetic segments with a thinned end-diastolic wall thickness, 15 (38%) achieved functional recovery, whereas dobutamine echocardiography detected recovery in only eight (53%). Further, of these 15 viable, wall-thinned segments, 12 (80%) showed an increased end-diastolic wall thickness after revascularization (mean ± SD were from 5.6 ± 0.7 mm at baseline to 7.4 ± 1.3 mm and 9.7 ± 1.4 mm after 1 week and after 3 months, respectively), and only 5 (42%) of these responded to dobutamine.

Conclusion: Dobutamine echocardiography showed a lessened sensitivity to predict post-revascularization functional recovery in akinetic/dyskinetic segments with a thinned end-diastolic wall thickness that subsequently increased in size.

Key Words: Dobutamine echocardiography, end-diastolic wall thickness, revascularization, chronic coronary artery disease, reversibility.

Introduction

Although a thinned end-diastolic wall thickness with absent systolic thickening is said to indicate non-viable myocardium in patients with chronic coronary artery disease [1], some investigators, on using positron emission tomography, have detected residual metabolic activity in certain chronic coronary artery disease patients with a thinned end-diastolic wall thickness and an absence of systolic wall thickening [2,3]. It thus may be that some akinetic myocardia with a thinned end-diastolic wall thickness may recover their systolic functions after coronary revascularization. However, there have been few reports on the functional outcomes of akinetic myocardial cases with a thinned end-diastolic wall thickness, and they have shown that most wall-thinned myocardia cannot recover [4,5]. There have been no previous reports on changes in thinned end-diastolic wall thickness after revascularization.

Dobutamine echocardiography has received considerable attention as a simple, safe, non-invasive, and relatively inexpensive method for diagnosing coronary artery disease. An experimental study [6] has shown that low doses of dobutamine may improve the contractile response of hypoperfused but viable myocardium with impaired systolic function. Even though dobutamine echocardiography is an established method to predict functional recovery after coronary revascularization in patients with chronic coronary artery disease [7-13], its accuracy is not always certain, especially in akinetic...
segments, since recent studies have demonstrated a lowered sensitivity in akinetic segments. Moreover, it is unknown whether dobutamine echocardiography can detect an akinetic but viable myocardium with a thinned end-diastolic wall thickness of the type mentioned above.

Therefore, to clarify these issues, this study was designed to evaluate the accuracy of dobutamine echocardiography in predicting post-revascularization functional recovery in myocardia considering both the varying degrees of asynergy, as well as, the serial changes in end-diastolic wall thickness in patients who have undergone coronary revascularization.

Methods

Study population

We screened 125 consecutive patients with chronic coronary artery disease undergoing coronary revascularization. Of these 125 patients, 63 were excluded for the following reasons: (1) acute myocardial infarction or unstable angina within the previous 6 weeks (12 patients); (2) absence of regional wall motion abnormalities that correlate with the site of the coronary stenosis on echocardiography (16 patients); (3) history of sustained ventricular tachycardia or atrial fibrillation (five patients); (4) any history that involved the use of beta-adrenergic blocker therapy (17 patients); (5) inadequate echocardiography for visualization due to a poor acoustic window (10 patients), and (6) patient refusal to participate (three patients). Of the remaining 62 patients, nine were withdrawn from the study because they were lost to follow-up study (five patients) or they underwent unsuccessful coronary angioplasty (four patients). Therefore, 53 patients constituted the final study population. The 45 men and eight women ranged in age from 41 to 77 years (mean 60 ± 3 years). Of the 53 patients, myocardial infarction had occurred in 43 patients from 6 to 432 weeks before. Further, 33 of these 43 patients had a Q wave old myocardial infarction and the remaining 10 had a non-Q wave old myocardial infarction. Of these 53 patients, 17 were asymptomatic, 28 had stable angina, four had atypical chest pain and four had dyspnoea on effort. All patients had at least one significant stenosis (≥75% of maximal luminal diameter) of a major epicardial coronary artery. Of the 53 patients, 29 had single-vessel, 19 had two-vessel, and five had three-vessel disease. Revascularization was achieved by percutaneous transluminal coronary angioplasty in 52 patients and coronary arterial bypass graft in one patient. The indications for coronary angioplasty were significant stenosis (≥75%) of any major coronary artery and provokable ischaemia by exercise tolerance test or ≥90% stenosis with a negative exercise tolerance test. Medication provided before and after revascularization was identical for each patient. The study was approved by the institutional ethical committee on clinical investigation, and informed consent was obtained from all patients.

Dobutamine echocardiography

Dobutamine echocardiography was performed approximately 1 week before coronary revascularization per a method previously described. In brief, all patients underwent two-dimensional echocardiography, using a Hewlett-Packard Sonos 1500 or Toshiba SSH-160A ultrasound system equipped with a 3.5 MHz or 2.5 MHz transducer, and parasternal long-axis, mid-ventricular parasternal short-axis, apical two- and four-chamber images were acquired. In addition to videotape recording, a digital on-line system with a cine loop presentation was simultaneously stored on a computer analysis system (Tomtec Imaging), with the Hewlett-Packard Sonos 1500 for the subsequent analysis. As to procedure, atropine sulfate (0.5 mg) was injected into a peripheral vein and echocardiography was repeated. Dobutamine was then administered intravenously at doses of 4, 8, 12, and 20 μg·kg⁻¹·min⁻¹ at 5 min intervals. Two-dimensional views were acquired during the last 1 min of each stage. The dobutamine infusion was stopped in the event of: (1) anginal pain, (2) a >15 mmHg decline in systolic blood pressure, (3) significant ventricular or supraventricular arrhythmia, (4) attainment of 80% of the maximal predicted heart rate, or (5) a new or worsened abnormality in systolic wall thickening in any segment. Follow-up echocardiographic views were obtained of all patients at 1 week and at 3 months after successful revascularization.

Analysis of segmental wall motion

Videotaped, echocardiographic images, obtained by the Toshiba SSH-160A, were stored in a Prism 5000 cine view operating system (Tomtec Imaging) that allows for the display of a continuous cine loop obtained from a single cardiac cycle and for side-by-side views of the left ventricle in a quad-screen format. All digitized images were independently assessed by two experienced observers who had no prior knowledge of the patients’ clinical or angiographic data. The left ventricle was divided into 16 segments per a method previously described. Segmental wall motion was graded as 1, normal; 2, hypokinesia; 3, akinesis; or 4, dyskinesia. In evaluating segmental wall motion abnormalities, systolic thickening in the central portion of each segment was carefully inspected. An improvement ≥1 in this segmental score, compared to its baseline value, during dobutamine infusion or after revascularization was considered significant. Inter- and intra-observer concurrence with regard to the assigned resting wall motion score was 92% (142/154) and 91% (140/154), respectively. Any discrepancies between the scoring of two observers were resolved by consensus after reevaluation of the images.
Echocardiographic analysis of end-diastolic wall thickness

For evaluations of end-diastolic wall thickness, qualitative and quantitative analyses were performed. Since apical cross-sections do not permit a reliable identification of endocardium[17], which is needed for measurement of wall thickness, we used parasternal long-axis or short-axis views, whenever possible, to evaluate wall thickness quantitatively. Apical long-axis view were only used for qualitative evaluation, since apical portions usually involved ischemia.

Qualitative analysis
Two independent observers visually evaluated all dysfunctioning segments, to determine whether the baseline end-diastolic wall thickness was reduced in comparison to normal segments. Of the 154 segments in which the baseline end-distolic wall thickness was qualitatively evaluated, a unanimous decision was reached in 149 (97%), and of the 154 segments in which end-diastolic wall thickness was reevaluated for change in wall thickness after revascularization, a unanimous decision was reached in 151 segments (98%); in the remaining segments, a split decision was resolved by consensus.

Quantitative analysis
The end-diastolic wall thickness of dysfunctioning segments was measured twice, in a parasternal long-axis view or a short-axis view before (pre-EDWT) and after revascularization (post-EDWT), at maximal ventricular cavity diameter by an independent observer. The mean of the two measurements was then considered to be the end-distolic wall thickness of that particular segment. Using video-taped images or digitized images in the cine-loop format, the thickness was measured at the centre of each region of interest by manually identifying a point on the epicardial and endocardial borders perpendicular to the two surfaces, and the length of the line joining these two points was then taken as being the wall thickness. Dysfunctioning segments were considered to have thinned if their pre-EDWT was <75% of the thickness of the adjacent normal segments[9]. An end-diastolic wall thickness was considered to have increased after revascularization if the post-EDWT value was ≥135% of the pre-EDWT value. This criterion was determined by assessing inter-observer and intra-observer differences in the end-diastolic wall thickness values. A coefficient of variation of 15% were present for both inter-observer and intra-observer variations. Thus, a 35% increase in the end-diastolic wall thickness value represents the 95% confidence level for detecting a significant change in the end-diastolic wall thickness value in our laboratory. End-diastolic wall thickness was considered to have thinned and to have increased, based on a quantitative analysis of a parasternal long-axis view or a short-axis view, and qualitative analysis of an apical long-axis view.

Coronary angiography and angioplasty

All of the patients underwent coronary angiography before and 3 months after revascularization. All angiograms were analysed qualitatively by an independent expert who had no knowledge of the echocardiographic data. If the follow-up angiography showed re-stenosis, a second coronary angioplasty was performed, if possible.

Statistical analysis

Either an analysis of variance or Student’s t-test was used to test for differences in the changes in the end-diastolic wall thickness among groups. Data are expressed as the mean ± standard deviation (mean ± SD). A P value <0.05 was considered significant.

Results

Coronary angiography and angioplasty

Coronary stenosis was 100% in 10 vessels, 99% stenosis with delayed flow in 12, 99% in three, 90% in 26, and 75% in eight vessels. As regards dilatation, 59 vessels in 53 patients were dilated, these vessels consisting of 36 left anterior descending, 14 right, and nine circumflex coronary arteries. Of these 59 vessels, 22 showed restenosis on follow-up angiography 3 months after coronary angioplasty and thus were given a second coronary angioplasty, and at 3 months after this second round of coronary angioplasty, stenosis was still present in 16 vessels.

Echocardiographic follow-ups

Left ventricular asynergy at baseline was observed in 174 myocardial segments. Twenty of these segments were excluded from this study, since each found to be related to restenosis of a coronary artery and had shown no improvement at either 1 week or 3 months after revascularization. Of the remaining 154 segments, 96 segments were judged hypokinetic, 42 were akinetic and 16 were dyskinetic. Of these 154 segments, 51 segments showed improved wall motion at 1 week after revascularization, and an additional 31 segments manifested wall motion recovery at 3 months after revascularization. Thus, a total of 82 segments exhibited improved regional wall motion after revascularization.

Dobutamine echocardiography

Changes in each segment after dobutamine infusion, and at 1 week and 3 months after revascularization are shown in Fig. 1. Of the 154 segments, dobutamine echocardiography identified a contractile reserve in 86. The results of dobutamine echocardiography and the post-revascularization functional recovery in each seg-
Predicting recovery in CAD

Wall motion

After dobutamine

Baseline

Follow-up post 1 week

Follow-up post 3 months

Normal

Hypokinesis

Akinesis

Dyskinesis

Table 1 Diagnostic accuracy of dobutamine echocardiography and the presence of a preserved end-diastolic wall thickness in the detection of post-revascularization functional recovery

<table>
<thead>
<tr>
<th>DE</th>
<th>Sensitivity</th>
<th>Specificity</th>
<th>Predictive accuracy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total (n=154)</td>
<td>81%</td>
<td>72%</td>
<td>76% 79%</td>
</tr>
<tr>
<td>Aki/dyskinesis (n=58)</td>
<td>69%</td>
<td>100%</td>
<td>100% 72%</td>
</tr>
<tr>
<td>Hypokinesis (n=90)</td>
<td>88%</td>
<td>56%</td>
<td>67% 79%</td>
</tr>
<tr>
<td>Preserved EDWT Aki/dyskinesis (n=58)</td>
<td>53%</td>
<td>92%</td>
<td>89% 62%</td>
</tr>
</tbody>
</table>

Aki/dyskinesis = Akinesis/dyskinesis, EDWT = end-diastolic wall thickness, DE = dobutamine echocardiography.

End-diastolic wall thickness and functional outcome

Of the 58 akinetic/dyskinetic segments, 39 had shown a thinned end-diastolic wall thickness at baseline before revascularization. All of the quantitatively measured, thinned segments were also considered thinned on qualitative analysis. Of these 39 segments with a thinned end-diastolic wall thickness, 15 (38%) showed improvement in wall motion after revascularization, and of the 19 akinetic/dyskinetic segments with a preserved end-diastolic wall thickness, 17 (89%) showed improved wall motion after revascularization. The diagnostic accuracy of the presence of a preserved end-diastolic wall thickness in akinetic/dyskinetic segments as methods to predict post-revascularization functional recovery is shown in Table 1. As for the presence of a preserved end-diastolic wall thickness in akinetic/dyskinetic segments, while the specificity (92%) and the positive predictive accuracy (89%) were high, the sensitivity (53%) and the negative predictive value (62%) were not.

End-diastolic wall thickness and dobutamine echocardiography

Figure 3 shows the dobutamine echocardiographic results and functional outcomes in akinetic/dyskinetic...
segments after revascularization, based on the presence or absence of end-diastolic wall thinning. Of 39 segments with a thinned end-diastolic wall thickness, eight manifested improved wall motion during dobutamine infusion and all eight achieved functional recovery after revascularization. Of 31 akinetic/dyskinetic segments with a thinned end-diastolic wall thickness that did not respond to dobutamine, seven false-negative segments achieved functional recovery after revascularization. Thus, of 15 segments with viable end-diastolic wall thinning, dobutamine detected only eight (53%). However, of 17 segments with a preserved end-diastolic wall thickness that achieved functional recovery after revascularization, dobutamine detected 14 (82%).

Only one hypokinetic segments had a thinned end-diastolic wall thickness, and though it showed no improvement during dobutamine infusion, wall motion did improve after revascularization.

Table 2 Serial changes in the end-diastolic wall thickness in hypokinetic segments, akinetic/dyskinetic segments, and segments with an increased end-diastolic wall thickness after revascularization

<table>
<thead>
<tr>
<th></th>
<th>Increased EDWT</th>
<th>Akinesis (n=18)</th>
<th>Hypokinesis (n=45)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n=8</td>
<td>RE(+) n=11</td>
<td>RE(-) n=7</td>
</tr>
<tr>
<td>Baseline (mm)</td>
<td>5.6±0.7</td>
<td>7.7±2.5</td>
<td>6.8±1.8</td>
</tr>
<tr>
<td>Follow up</td>
<td>7.4±1.3*</td>
<td>8.8±2.0</td>
<td>6.6±1.9</td>
</tr>
<tr>
<td>1 week (mm)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3 months (mm)</td>
<td>9.7±1.4*</td>
<td>9.3±2.4*</td>
<td>6.7±1.6</td>
</tr>
</tbody>
</table>

EDWT=end-diastolic wall thickness, RE=Reversibility.

*P<0.05 vs baseline by ANOVA.
Predicting recovery in CAD

Figure 4  End-diastolic (upper panels) and end-systolic (lower panels) midventricular short-axis views under basal conditions (base) and during 4 to 12 μg·kg⁻¹·min⁻¹ of infused dobutamine. No improvement in systolic thickening was observed during dobutamine infusion in posterior segments, which were akinetic with end-diastolic wall thinning at baseline (arrowhead).

Figure 5  Same patient as in Fig. 4. Upper panels show end-diastolic midventricular short axis views before coronary angioplasty (Pre PTCA) and at 1 week (Post PTCA 1 Week) and 3 months (Post PTCA 3 Months) after successful PTCA. Lower panels show same views at end systole. Improvement in systolic thickening was observed after PTCA in posterior segment with thinned end-diastolic wall thickness in baseline (5.2 mm, arrowhead), which increased at 1 week (7.7 mm) and at 3 months (10 mm) after PTCA.

about half of these segments. Further, most of these segments had an increased end-diastolic wall thickness after revascularization.

**Dobutamine echocardiography**

In a previous report we have demonstrated that dobutamine echocardiography possesses excellent sensitivity and specificity for identifying hibernating myocardium. As regards the merits of dobutamine echocardiography in assessing myocardial viability, most of the previous dobutamine echocardiographic studies have evaluated only dysfunctioning segments, whereas the present study evaluated various degrees of asynergy in which the sensitivity and specificity of dobutamine echocardiography were, respectively, 69% and 100% in akinetic/dyskinetic segments, and 86% and 56% in hypokinetic segments. These results have indicated that while there were a few false-positive cases among akinetic/dyskinetic segments, there were many false-negative cases too, as well as many false-positive cases in hypokinetic segments.

Two recent studies have reported on problems with dobutamine echocardiography in akinetic segments. Arnese et al. have found that dobutamine echocardiography detected myocardial viability more frequently in severe hypokinesis than in akinesis. Perrone-Filardi et al. have reported that the overall sensitivity and specificity for identifying dysfunctioning segments were 88% and 87%, respectively, and for identifying akinetic segments, 69% and 100%, respectively. These findings are similar to the findings of our study, although lower doses of dobutamine were used and coronary angiography was not performed at follow-up.

La Canna et al. have reported that sensitivity in akinetic segments after coronary artery bypass graft was 88-8% and that the specificity was 81-6%. This higher
sensitivity, in contrast to the findings of our study, has several possible explanations. The patients of their study may have had less severe coronary stenosis, which did not trigger dobutamine-induced ischaemia; they also had greater end-diastolic wall thickness baseline values (mean: 10 ± 4 mm) than the patients of our study (mean: 7.7 ± 2.5 mm). Also, failure to assess graft closure might have led to underestimating the true number of viable segments, which would have resulted in higher sensitivity and lower specificity findings. One explanation that may account for a decrease in specificity in akinetic/dyskinetic segments may be ‘reverse tethering’ that can occur during dobutamine infusion, wherein non-viable segments give the appearance of being active due to the contraction of adjacent normal segments. To eliminate this possibility, our study evaluated wall motion in the central portion of each segment.

In hypokinetic segments, the high incidence of false-positive cases might have been due to the dobutamine infusion, which may improve the function of residual viable myocardium in segments with non-transmural infarction. An experimental study has found that the absolute extent of wall thickening by dobutamine during reflow is directly related to the amount of myocardium that has escaped necrosis. Further, a hypokinetic myocardium indicates the presence of a still viable myocardium, irrespective of post-revascularization functional recovery. Therefore, any improvement in a hypokinetic myocardium by dobutamine cannot be used to predict functional recovery after revascularization.

End-diastolic wall thickness and functional outcome

Pathological studies have revealed that a completely scarred myocardium of chronic infarcts is commonly associated with substantial myocardial thinning to <6 mm20 or even with aneurysmal formation, and that complete myocardial healing in humans takes 6 to 8 weeks.21,22 However, the present study found that, in patients with old myocardial infarction, some akinetic segments with a thinned end-diastolic wall thickness showed improved wall motion after coronary revascularization. This finding is bolstered by a report that has identified residual metabolic activity in the form of glycolysis in myocardial regions with reduced end-diastolic wall thickness and absent systolic wall thickening.22

A recent study has reported that the presence of a preserved end-diastolic wall thickness was predictive of postoperative recovery in 18 of 25 (72%) akinetic segments, and the presence of scar tissue as a reduced end-diastolic wall thickness and an abnormal increase in acoustic reflectance was predictive of an irreversibly damaged myocardium in 13 of 15 segments (87%).9 Although our high positive predictive value in the presence of a preserved end-diastolic wall thickness for predicting post-revascularization functional recovery agreed with the former result (72%), our lower negative predictive value compared with the latter result (87%) requires conjecture. One possibility is that their study considered scar tissue as a reduced end-diastolic wall thickness and an abnormal increase in acoustic reflectance, which meant that it included a high percentage of irreversible damaged tissue. In fact, although our study avoided showing the result of acoustic reflectance because of its subjective methodology, none of our viable wall-thinned myocardium showed abnormal acoustic reflectance. A second possibility may be that their patients underwent revascularization ≥12 weeks after acute myocardial infarction whereas our patients underwent revascularization ≥6 weeks after acute myocardial infarction. This earlier revascularization may have resulted in a higher percentage of viable wall-thinned myocardium in our study.

Dobutamine echocardiography and ‘Reversible wall-thinned myocardium’

Based on our findings, in akinetic/dyskinetic segments with a preserved end-diastolic wall thickness, dobutamine echocardiography was able to detect 84% of the segments that showed functional recovery after revascularization. In contrast, dobutamine improved contractility in only 53% of the segments with viable end-diastolic wall thinning. All of the segments that did not respond to dobutamine and had a viable end-diastolic wall thinning showed an increased end-diastolic wall thickness after revascularization.

That some of our chronic coronary artery disease patients were found to have what we have termed a ‘reversible wall-thinned myocardium’ was the most striking findings of this study. Since no previous study has investigated serial changes in end-diastolic wall thickness after revascularization, and as there has been no previous evidence that a thinned end-diastolic wall thickness undergoes an increase and recovers its systolic functions after revascularization in some chronic coronary artery disease patients, the anatomy and physiology of such segments have yet to be investigated. However, there are some mechanisms that may account for this end-diastolic wall thickness reversal. First, the vascular volume decreases as a result of coronary artery disease, thereby contributing to wall thinning, and after revascularization, the vascular volume again increases. In fact, giving support to this speculation, the coronary stenosis present in these segments was found to be very severe. In this regard, Vogel et al.23 noted that the loss of vascular filling in acute global ischaemia can account for reductions in end-diastolic wall thickness of up to 18%.23 Second, it has been reported that the stretching that occurs in individual myocytes may result in myocytic lengthening and thinning due to wall stress.24 Finally, a loss of contractile material24 may produce a rearrangement of myocytes that results in cell slippage. As the recovery of function through the regeneration of contractile material requires a considerable amount of
time, this time element is consistent with the findings of the present study, in which a greater increase in wall thickness was seen at 3 months after revascularization than at 1 week after revascularization.

These mechanisms that may account for a 'reversly wall-thinned myocardium' may also reduce the contractile response to dobutamine. Further, since the coronary stenosis seen in these segments was much more severe, these segments might have exhausted the coronary reserve, and may only retain a subepicardial but not subendocardial coronary reserve. Therefore, inotropic stimulation might not elicit a contractile response in these segments despite the presence of viable myocardium. Future investigations should be done in increased numbers of such segments, and focus on studying ultrastructural anatomy of these segments and on alternate modalities to identify them other than dobutamine echocardiography.

Limitations

The limitations of this study must be considered. First, for evaluations of end-diastolic wall thickness, qualitative and quantitative analyses were performed. Although qualitative analysis was not time-consuming, showed good reproducibility, and accord with the quantitative one, future investigations should be done with alternate modalities to evaluate end-diastolic wall thickness quantitatively other than echocardiography. Second, the effect of volume on end-diastolic wall thickness was not investigated. Third, it should be pointed out that a hibernating myocardium may remain 'stunned' for up to 1 year or longer after revascularization. However, follow-up echocardiography was performed at 3 months after revascularization. The effect of this early follow-up echocardiography was minimal because most of the hibernating myocardium had recovered by that time. Finally, since most of our patients had a single-vessel disease and only a few had a greatly depressed global systolic function, it is necessary to investigate more of the latter patients before any conclusions can be reached.

Conclusions

The major findings in this study were that some chronic coronary artery disease patients with akinetic/dyskinetic segments and a thinned end-diastolic wall thickness achieved functional recovery and showed an increase in the end-diastolic wall thickness after revascularization, and that the use of dobutamine echocardiography for predicting functional recovery after revascularization resulted in too many false-positive cases in the hypokinetic segments, although the specificity of dobutamine echocardiography for assessing akinetic/dyskinetic segments was excellent. However, dobutamine echocardiography did not show as a high sensitivity for assessing akinetic/dyskinetic segments with a thinned end-diastolic wall thickness.

References


