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Assessment of Dyssynchronous Wall Motion During Acute Myocardial Ischemia Using Velocity Vector Imaging

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OBJECTIVES The purpose of this study was to investigate the diagnostic value of velocity vector imaging (VVI) for detecting acute myocardial ischemia and whether VVI can accurately demonstrate the spatial extent of ischemic risk area.

BACKGROUND Using a tracking algorithm, VVI can display velocity vectors of regional wall motion overlaid onto the B-mode image and allows the quantitative assessment of myocardial mechanics. However, its efficacy for diagnosing myocardial ischemia has not been evaluated.

METHODS In 18 dogs with flow-limiting stenosis and/or total occlusion of the coronary artery, peak systolic radial velocity (V_{SYS}), radial velocity at mitral valve opening (V_{MVO}), peak systolic radial strain, and the percent change in wall thickening (%WT) were measured in the normal and risk areas and compared to those at baseline. Sensitivity and specificity for detecting the stenosis and occlusion were analyzed in each parameter. The area of inward velocity vectors at mitral valve opening (MVO) detected by VVI was compared to the risk area derived from real-time myocardial contrast echocardiography (MCE). Twelve image clips were randomly selected from the baseline, stenosis, and occlusions to determine the intra- and inter-observer agreement for the VVI parameters.

RESULTS The left circumflex coronary flow was reduced by 44.3 ± 9.0% during stenosis and completely interrupted during occlusion. During coronary artery occlusion, inward motion at MVO was observed in the risk area. Percent WT, peak systolic radial strain, V_{SYS}, and V_{MVO} changed significantly from values at baseline. During stenosis, %WT, peak systolic radial strain, and V_{SYS} did not differ from those at baseline; however, V_{MVO} was significantly increased (−0.12 ± 0.60 cm/s vs. −0.96 ± 0.55 cm/s, $p = 0.015$). Sensitivity and specificity of V_{MVO} for detecting ischemia were superior to those of other parameters. The spatial extent of inward velocity vectors at MVO correlated well with that of the risk area derived from MCE ($r = 0.74$, $p < 0.001$ with a linear regression).

CONCLUSIONS The assessment of VVI at MVO permits easy detection of dyssynchronous wall motion during acute myocardial ischemia that cannot be diagnosed by conventional measurement of systolic wall thickness. The spatial extent of inward motion at MVO suggests the size of the risk area. (J Am Coll Cardiol Img 2008;1:210–20) © 2008 by the American College of Cardiology Foundation

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The assessment of regional wall motion abnormalities in left ventricular (LV) myocardium is necessary for diagnosing ischemic heart disease. In particular, post-systolic thickening or shortening, which is defined as myocardial contraction after aortic valve closure (AVC), has been noted as a highly sensitive marker of myocardial ischemia. The analysis of regional myocardial velocity, strain rate, or strain assessed using the tissue Doppler technique permits sensitive detection of post-systolic thickening and improves the accuracy of diagnosis of ischemic heart disease (1–4). However, the angle dependency of the Doppler technique frequently hampers the evaluation of regional wall motion abnormalities, including post-systolic thickening, in ischemic myocardium (5–7).

B-mode tissue tracking is a promising method for evaluating regional wall motion without angle dependency (8–10). Using a novel feature-tracking algorithm, velocity vector imaging (VVI) can display velocity vectors of regional wall motion overlaid onto the B-mode image and allows the quantitative assessment of LV myocardial mechanics (11,12). We have already reported that abnormal inward wall motion, which is presumed to be due to post-systolic thickening, can be detected in the latter half of isovolumic relaxation during myocardial ischemia using VVI (13). However, its efficacy for diagnosing myocardial ischemia has not been elucidated. Because dyssynchronous wall motion of LV myocardium can be easily visualized using velocity vectors, we hypothesized that VVI would allow sensitive detection of dyssynchrony induced by post-systolic thickening during ischemia and be able to demonstrate the accurate spatial extent of the ischemic risk area without angle dependency.

In this study, we investigated the diagnostic value of VVI for detecting the critical state of acute myocardial ischemia by comparing it with the conventional measurement of systolic wall thickening in anesthetized dogs. We also evaluated whether the spatial extent of post-systolic inward motion detected by VVI corresponds with that of ischemic myocardium indicated by myocardial contrast echocardiography (MCE).

METHODS

Animal preparation. All animal studies were performed in accordance with guidelines for the care and use of laboratory animals at our institution. Eighteen open-chest dogs were used in this study. Dogs (weighing 14.1 ± 0.6 kg) were anesthetized using intravenous pentobarbital sodium (35 mg/kg), intubated, and ventilated with room air using a respirator pump. An 18-gauge peripheral intravenous catheter positioned in the foreleg was used for administration of fluids, drugs, and contrast microbubbles during MCE. Anesthesia with pentobarbital sodium was maintained throughout the experiment (6 to 8 mg/kg/h). A 5-F catheter was placed in the ascending aorta to monitor blood pressure, and the electrocardiogram was monitored continuously.

Dogs were placed in the right recumbent position. A left lateral thoracotomy was performed, and the heart was suspended in a pericardial cradle. The proximal portion of the left circumflex artery (LCx) and/or left anterior descending artery (LAD) was dissected free from surrounding tissues, and a vascular occluder was placed to create a flow-limiting stenosis or total occlusion. A perivascular ultrasonic flow probe was placed at the distal site of the occluder and connected to a digital flowmeter (Transonic Systems, Ithaca, New York). Flow-limiting stenosis was set to be approximately half of the baseline flow, in which systolic wall thickening is relatively preserved (14).

Echocardiography. VVI. Echocardiography was performed using a Sequoia ultrasound system (Siemens Medical Solutions, Mountain View, California). The LV short-axis view at the papillary muscle level was visualized using a water bath as a standoff. The position of the ultrasound transducer for the short-axis view was fixed with a mechanical arm. Two-dimensional images (transmitting and receiving frequencies 2.0 and 4.0 MHz, respectively) for regional wall motion assessment were captured over 3 consecutive cardiac cycles. The frame rate was set at 80 to 84 frames/s. For the detailed evaluation of regional wall motion, the timing of mitral valve opening (MVO) was determined by measurement of mitral inflow in the apical 4-chamber view by pulse Doppler (2.0 MHz) and that of AVC was assessed from the aortic component of the second heart sound derived from the phonocardiogram, which was simultaneously displayed with Doppler data. When the apical view was scanned, the transducer was removed from the mechanical arm and held manually. A microphone for the phonocardiogram was placed directly on the base of the aorta. Data were digitally stored on magneto-optical disks for subsequent off-line analysis.

High frame rate acoustic capture B-mode data were analyzed using off-line software (Syngo Ve-

ABBREVIATIONS AND ACRONYMS

AVC = aortic valve closure
CI = confidence interval
LAD = left anterior descending artery
LCx = left circumflex artery
LV = left ventricule/ventricular
MBF = myocardial blood flow
MCE = myocardial contrast echocardiography
MVO = mitral valve opening
ROC = receiver operating characteristic
V_{MVO} = radial velocity at mitral valve opening
V_{SYS} = peak systolic radial velocity
VVI = velocity vector imaging
WT = wall thickening