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Assessment of Left Ventricular Function in Symptomatic Patients with Myocardial Bridge using Two-Dimensional Strain

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ABSTRACT

Background and Objectives : We wanted to perform quantitative echocardiographic assessment of myocardial function in the patients with myocardial bridge by measuring 2-dimensional strain with using newly developed software. **Subjects and Methods :** Novel computer software was used for tracking heart tissue on echocardiography, and we conducted an advanced wall-motion analysis for 18 symptomatic patients (mean age: 57.1±9.7 years, 10 female) with myocardial bridging of the left anterior descending coronary artery and also 20 age-matched healthy controls. The conventional wall-motion scoring was normal in all the patients, and the software was able to adequately track their heart tissue. **Results :** The maximal angiographic systolic lumen diameter reduction within the myocardial bridges was 71±12.6% at rest, with a persistent diameter reduction of 31.2±11.3%. The radial strain and displacement of the anterior segments were more significantly reduced than that of the posterior segments at the level of the papillary muscle (30.9±13.8% vs. 51.8±17.3% and 4.8±0.9 vs. 5.9±1.5, respectively, all $p<0.05$), and this showed a plateau (39% and 33%, respectively) or biphasic (50% and 56%, respectively) pattern. The time from the R wave on electrocardiography to the transition from regional systole to early diastolic lengthening (Tr) was significantly delayed in the patients with myocardial bridge more than that for the controls (497±20.4 ms vs. 348±12.5 ms, respectively, $p<0.05$). **Conclusion :** Delayed systolic contraction and diastolic relaxation are important mechanisms that contribute to ischemia in the patients with myocardial bridge. 2-dimensional strain can be used to achieve real-time wall-motion analysis, and it has the potential to improve the identification and functional quantification of myocardial Bridge. **(Korean Circulation J 2006;36:617-625)**

KEY WORDS : Echocardiography ; Strains.

Introduction

Although myocardial bridges (MB) are commonly found in normal human hearts,[1)2)] their association with anginal chest pain or abnormal results on noninvasive tests constitutes an important clinical issue. Myocardial bridges have been associated with stable and variant angina, sudden death and myocardial infarction,[1-7)] a variety of coronary syndromes with twofold levels of disturbed coronary hemodynamics[8)] and the endothelial dysfunction[9)] that is caused by extra vascular vessel compression. The mechanism of this ischemia has recently been clarified, and it consists of a significant reduction of the diastolic coronary diameter, in addition to the phasic systolic compression.

Traditionally, the regional myocardial function has been assessed by conventional wall-motion analysis and by such methods of quantitative echocardiography as integrated backscatter, automatic border detection and tissue Doppler imaging (TDI). We present here a novel method for the real-time quantitative assessment of myocardial function on the basis of newly developed software for performing quantitative echocardiography, which allows a rapid, accurate and simple determination of the global and regional myocardial parameters, i.e., velocity, strain and the strain rate. We evaluated the feasibility of determining the 2 dimensional strain in control subjects and in symptomatic patients with myocardial bridge (MB).

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Subjects and Methods

Study population

We studied 18 patients who suffered with angina; the

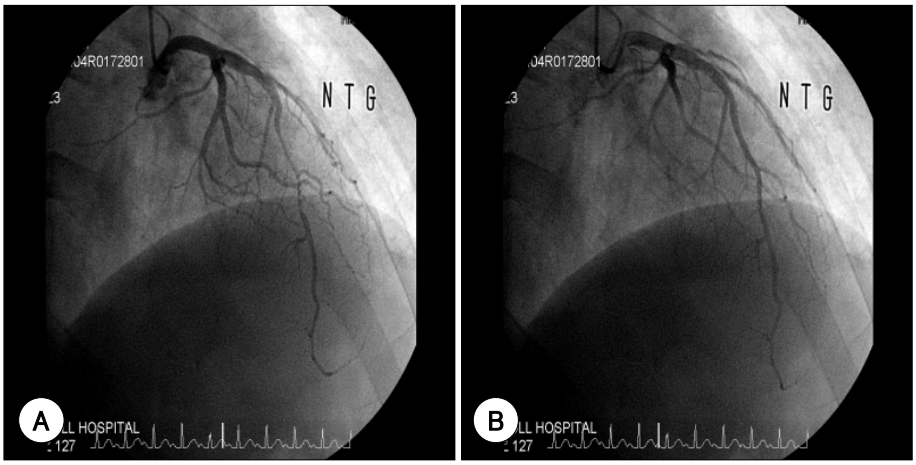

Fig. 1. Angiographic images of the left anterior descending coronary (LAD) artery revealed pronounced compression in the middle of the LAD artery during systole (A) and no compression of the LAD artery during diastole (B).

only relevant angiographic finding for these patients was compression of the mid-segment of the left anterior coronary artery by a MB, which caused a $\geq$50% reduction in the systolic vessel diameter, as was visually assessed (Fig. 1). All patients had a normal conventional wall-motion score based on the standards of the American Society of Echocardiography. The control group included 20 age-matched healthy individuals with normal results on echocardiography and exercise stress testing. 13 patients showed typical angina and 3 patients showed exercise-induced ST-segment depression, positive myocardial scintigraphy was noted in 8 patients and abnormal intracoronary Doppler flow velocities were noted in 4 patients; these findings are all recognized manifestations of ischemia. As a rule, the patients who didn't undergoing stress testing before cardiac catheterization had unstable symptoms (4 patients). None of the patients presented with concomitant coronary stenosis or any present or previous cardiac events. Echocardiography was done to rule out concomitant hypertrophic cardiomyopathy in all patients. An informed consent for the catheterization and physiologic assessment was obtained from all the patients. The demographic data was recorded, including age, gender and the cardiovascular risk factors.

Coronary angiogram

A biplane cardio-vascular system with a charge coupled device (CCD) (INTEGRIS BV 5000, Philips Medical System, Netherlands) was used for the digital imaging to determine the appropriate intervention and diagnosis and to visualize the left anterior descending coronary artery. The quantitative measurements were analyzed by a workstation with dedicated software (WIN 32 version 3.3). For this purpose, the coronary segment of interest was identified, including the total length of the myocardial bridge with the adjacent non-obstructed proximal and distal segments. The boundaries of this segment were then detected automatically and corrected manually if necessary. The absolute vessel diameters (mm) were determined with using the guiding catheter as a reference. The percent diameter stenosis at the most severe site was automatically calculated from the computer estimation of the original dimension of the artery along the myocardial bridge; this was defined by interpolation between the proximal and distal reference diameters. Evaluation of the angiographic lumen diameter was performed at the end of systole, when the diameter of the vessel was smallest, and during the mid-diastolic frames. To determine the interobserver variability for the quantitative angiographic measurements of the systolic and diastolic lumen diameter reductions, images from a random subset of 18 patients were analyzed by two independent observers (K.T.I. and P.J.H.), who were blinded to each other's results.

Echocardiographic evaluation

A standard 2D and strain echocardiographic examination was performed on all subjects with them lying in the left lateral supine position and with using a 2.5-MHz transducer on Vivid 7 Dimension ultrasound equipment (General Electric, Horten, Norway). Images from the parasternal short axis views at the 3 levels (the mitral valve, papillary muscle and apex), the apical 4-chamber view, the 3-chamber (apical long axis) view and the 2-chamber view of the left ventricle (LV) were obtained at end-expiratory apnea, and these were stored in a cine-loop format for the subsequent offline analysis. Three heartbeats were collected from each view and then analyzed off-line with an EchoPAC Dimension system (General Electric, Horten, Norway). The LV was divided into 6 segments (anterior, inferior, lateral, posterior, septal and anteroseptal), and each of these was then subdivided into 3 segments (basal, medial and apical). The

myocardial displacement and systolic strain for each segment were calculated with the new tissue tracking software and these results were compared with the results obtained in the same segments with using conventional TDI. The frame rate for the TDI-based strain and the 2-D strain was 84 to 106 frames/s and 60 frames/s, respectively.

Selection of the representative tissue patterns

2-Dimensional strain was not based on the TDI and so we used frame-by-frame image tracking to estimate the tissue movement. This is an accepted approach for reconstruction of temporal movements and deformations. We have observed that ultrasound tissue images contain many small elements and natural acoustic markers, which move together with the tissue and they do not significan-

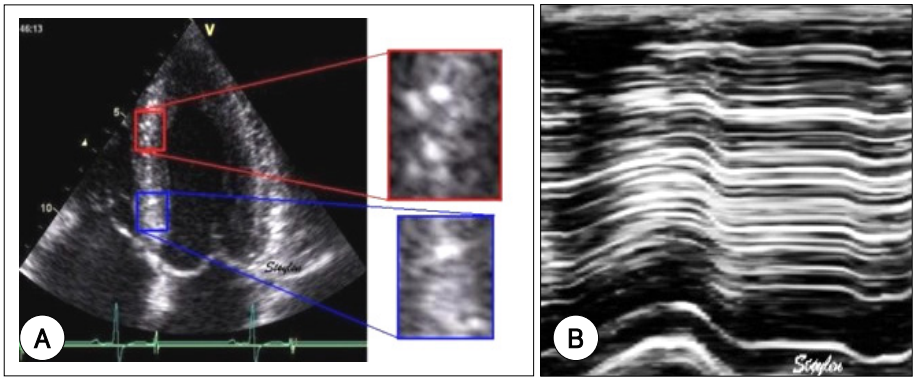

Fig. 2. Typical speckle patterns in the myocardium. The two enlarged areas show completely different speckle patterns, which creates a unique pattern for any selected region that can identify this region and hence, the displacement of the region in the next frame (A). When the speckle pattern is followed by an M-mode in the wall, the alternating bright and dark points are seen as alternating bright and dark lines, and it shows true myocardial motion (B). Adapted from the strain rate imaging by Asbjørn Støylen MD. with the permission of the publisher.

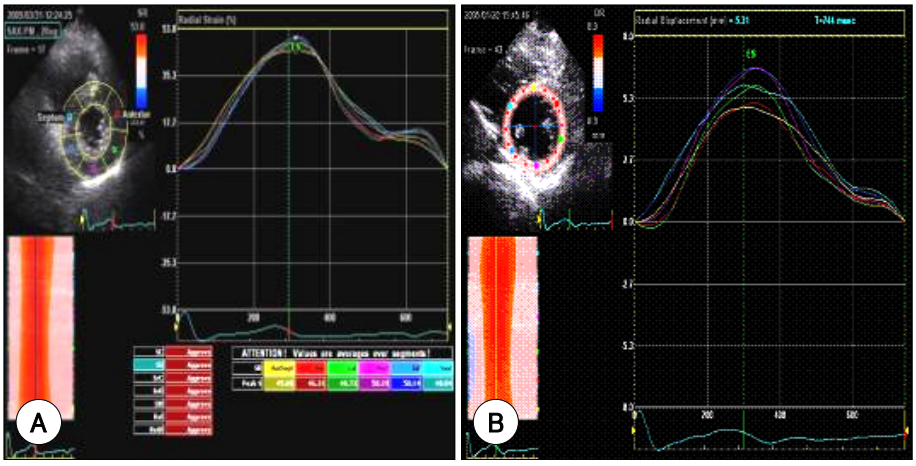

Fig. 3. 2D strain images obtained from the control. Parasternal short axis view. Radial strain (A) and displacement (B) from the papillary muscle level show uniformity through the segments. 2D: 2-dimensional.

Table 1. Demographic and clinical characteristics

	Myocardial bridge (n=18)	Control (n=20)
Age (years)	57.1±9.7 (43-74)	54.6±8.6 (38-75)
Gender (M/F)	8/10	10/10
Interval symptoms/diagnosis (months)	8.3±9.5 (0-44)	0
No. of hospital admissions	1.8±0.7 (1-8)	0
Angina pectoris (overall/typical/atypical) (%)	18 (100%)/13 (72%)/5 (28%)	0
Positive exercise testing	3 (17%)	0
Acute coronary syndrome	4 (22%)	0
Hypertension	3 (17%)	0
Ejection fraction (%)	66.7±3.9	67.8±9.5
Left ventricular mass index (g/m^2)	103.0±27.2	100.1±12.7
Relative wall thickness	0.41±0.03	0.39±0.7

Values are means±SD

tly change their pattern between the adjacent frames. These natural acoustic markers are stable acoustic speckles, and they are statistically equally distributed within the myocardium (Fig. 2A). Each marker can be identified and accurately followed during several consecutive frames (Fig. 2B). Despite their speckled structure, these elements are temporally stable. A spatial/temporal image processing algorithm was used, which allows recognition and selection of such elements on the ultrasound image. The geometric position of each marker changes from frame to frame in accordance with the surrounding tissue motion. The algorithm allows tracking of the new marker location on the sequential images with using correlation criteria and the sum of the absolute differences. The geometric shift of each element represents local tissue movement. The relative changes of the mutual distance between neighboring elements reflect the tissue's contraction/relaxation (the 2D strain rate and strain). The software was dependant on high-resolution image quality and it was applied with the harmonic imaging.

Statistical analysis

All the data are expressed as means±standard deviations (SD). The data were analyzed using standard statistical software (SPSS package version 11.0) and comparisons of all the measurements were made with paired Student's t-tests for the continuous variables and chi-square tests for the categorical variables. A p of <0.05 was considered to indicate significance.

Results

General characteristics of patients

The subjects' mean age was 57.1±9.7 years (range: 43-74 years). All the patients reported a history of chest pain that was strongly suggestive of angina pectoris. The mean time interval between their first symptoms and the angiographic diagnosis of a myocardial muscle bridge was 8.3±9.5 months. Three patients had a history of mild hypertension without any echocardiographic evidence of left ventricular hypertrophy. The global left ventricular function was normal in all the patients with myocardial bridges and there were no signs of left ventricular hypertrophy or hypertrophied cardiomyopathy. The major demographic and clinical characteristics of the subjects are given in Table 1.

Coronary angiographic finding

The complete quantitative coronary angiographic data are presented in Table 2. The mean percent diameter reduction at the most severe site of the myocardial bridge during systole was 71.0±12.6%, and this ranged from 51% to 95%. There was a persistent diastolic lumen diameter reduction of 31.2±11.3% with a minimum of 24% and a maximum of 58%. The mean absolute systolic diameter within the myocardial bridge was 0.7±0.3 mm; five patients had a minimal diameter <0.5 mm. The length of the myocardial bridge during systole ranged from 19 to 44 mm (mean length: 26±7 mm), and this was not different from the diastolic values of 24±6 mm (range: 21 to 42 mm, p=NS).

Table 2. Coronary angiographic measurements in 18 patients with myocardial bridge

	Coronary diameter (mm)
Proximal to MB (mm)	
Systolic	2.96±0.89
Diastolic	3.21±0.55
Distal to MB (mm)	
Systolic	2.50±0.42
Diastolic	2.60±0.41
With in MB (mm)	
Systolic	0.92±0.48
Diastolic	2.19±0.41
Length of MB (mm)	
Systolic	26±7
Diastolic	24±6
% diameter reduction	
Systolic	71.0±12.6%
Diastolic	31.2±11.3%

Values are means±SD. MB: myocardial bridge

Table 3. Comparison of regional strain with using 2DS and TDI between the patients with myocardial bridge (MB) and the controls at the 4-chamber views

4 chamberview	Variables	Control (n=20)	MB (n=18)	p
Basal septum	Strain TDI	-18.2±6.2	-17.9±5.8	NS
	TS (2DS)	42.3±9.7	41.5±10.1	NS
	LS (2DS)	16.5±2.6	16.7±1.2	NS
Mid septum	Strain TDI	-21.3±6.4	-20.2±5.1	NS
	TS (2DS)	24.8±5.3	23.2±8.7	NS
	LS (2DS)	21.6±4.2	20.9±3.9	NS
Apical septum	Strain TDI	-17.9±8.2	-18.8±7.8	NS
	TS (2DS)	24.8±9.6	25.1±11.2	NS
	LS (2DS)	25.3±6.8	24.2±7.1	NS
Apical lateral	Strain TDI	-15.2±7.3	-14.5±8.7	NS
	TS (2DS)	44.6±15.8	43.5±22.1	NS
	LS (2DS)	19.5±6.9	18.4±7.5	NS
Mid lateral	Strain TDI	-16.8±5.3	-17.2±4.5	NS
	TS (2DS)	47.2±24.3	46.8±28.7	NS
	LS (2DS)	18.5±6.2	17.6±5.9	NS
Basal lateral	Strain TDI	-16.2±4.8	-15.3±4.5	NS
	TS (2DS)	53.2±21.8	50.4±31.8	NS
	LS (2DS)	17.4±4.7	18.4±5.1	NS

Values are means±SD. 2DS: 2-dimensional strain, TDI: tissue Doppler imaging, 4ch: 4 chamber view, TS: transverse strain of 2DS, LS: longitudinal strain of 2DS, NS: not significant

Strain echocardiographic findings

Using the current software, the systolic strain and strain rate in the control group were relatively uniform

throughout the LV; these values were highest in the inferior segments and lowest in the anteroseptal segments, but the difference was not significant.

Table 4. Comparison of regional strain with using 2DS and TDI between the patients with myocardial bridge (MB) and the controls at the 2-chamber views

2 chamber view	Variables	Control (n=20)	MB (n=18)	p
Basal inferior	Strain TDI	-19.2±6.5	-19.0±5.3	NS
	TS (2DS)	41.3±8.7	42.8±11.8	NS
	LS (2DS)	17.5±5.6	19.7±1.2	NS
Mid inferior	Strain TDI	-20.3±4.4	-19.5±6.2	NS
	TS (2DS)	25.8±5.8	27.2±6.7	NS
	LS (2DS)	21.3±4.7	20.7±7.5	NS
Apical inferior	Strain TDI	-19.9±8.9	-22.1±9.5	NS
	TS (2DS)	25.8±9.5	24.1±10.2	NS
	LS (2DS)	23.3±5.8	21.2±5.1	NS
Apical anterior	Strain TDI	-18.2±7.7	-14.9±4.4*	0.04
	TS (2DS)	43.6±15.8	39.5±21.5	NS
	LS (2DS)	19.5±6.7	13.4±7.3*	0.01
Mid anterior	Strain TDI	-22.8±8.7	-18.4±6.3	<0.01
	TS (2DS)	48.2±24.3	37.8±24.3	<0.01
	LS (2DS)	20.5±6.8	15.6±3.5	0.02
Basal anterior	Strain TDI	-16.2±4.8	-17.0±7.2	NS
	TS (2DS)	51.2±20.7	49.5±29.6	NS
	LS (2DS)	15.4±4.9	14.3±6.1	NS

Values are means±SD. *:p<0.05 vs. inferior. 2DS: 2-dimensional strain, TDI: tissue Doppler imaging, 2ch: 2 chamber view, TS: transverse strain of 2DS, LS: longitudinal strain of 2DS, NS: not significant

Table 5. Comparison of regional strain with using 2DS and TDI between the patients with myocardial Bridge (MB) and the controls at the 3-chamber views

3 chamber view	Variables	Control (n=20)	MB (n=18)	p
Basal posterior	Strain TDI	-17.8±8.9	-16.7±4.4	NS
	TS (2DS)	41.3±12.7	38.5±15.1	NS
	LS (2DS)	16.3±1.6	17.7±1.9	NS
Mid posterior	Strain TDI	-20.3±4.8	-16.9±5.2	NS
	TS (2DS)	24.9±5.7	27.2±6.3	NS
	LS (2DS)	16.6±4.8	14.9±4.9	NS
Apical posterior	Strain TDI	-16.9±8.5	-17.1±6.0	NS
	TS (2DS)	22.8±8.5	24.1±9.2	NS
	LS (2DS)	20.3±6.2	18.2±5.7	NS
Apical ant.septum	Strain TDI	-14.2±7.3	-12.8±4.9*	NS
	TS (2DS)	43.6±15.8	48.4±28.0	NS
	LS (2DS)	15.5±6.8	13.4±7.5	NS
Mid ant.septum	Strain TDI	-21.8±5.3	-17.1±5.5	<0.01
	TS (2DS)	52.2±24.3	44.5±23.7	<0.01
	LS (2DS)	27.5±6.2	17.6±5.9	<0.01
Basal ant.septum	Strain TDI	-16.2±4.8	-13.6±4.3	NS
	TS (2DS)	52.3±21.9	50.4±31.8	NS
	LS (2DS)	16.7±4.5	13.4±6.1	NS

Values are means±SD. *: p<0.05 vs. posterior segment. 2DS: 2-dimensional strain, TDI: tissue Doppler imaging, 3ch: 3 chamber view, TS: transverse strain of 2DS, LS: longitudinal strain of 2DS. ant.: anterior

Regional strain obtained with 2D strain and TDI at the apical view

The mean comparative data for the novel software and TDI in the 18 patients with myocardial bridge (MB) are presented in Table 3, Table 4 and Table 5. The peak strains on the TDI were comparable with the longitudinal strains of the 2 dimensional strain, but the transverse strains were higher than the longitudinal strain. The peak longitudinal strain and TDI strain of the apical anterior and anteroseptal segments of the MB were significantly lower than that for the inferior and posterior segments of the MB and the controls (p<0.005). The peak transverse strains were relatively uniform throughout the LV.

Myocardial displacement and strain by the 2D Strain on the parasternal short axis views

The radial strain of the anteroseptal and anterior segments of the MB were significantly reduced more than that of the posterior and inferior segments of the MB and the controls at the level of mitral valve and mid ventricle (Table 6). The radial strain and displacement of the anterior segments of the MB were significantly reduced more than that of the posterior segments at the mid ventricle level (30.9±13.8% vs. 51.8±17.3% and 4.8±0.9 vs. 5.9±1.5, respectively, all p<0.05), and these features showed plateau (39% and 33%, respectively) or biphasic (50% and 56%, respectively) patterns (Table 7) (Fig. 3, 4). The time from the R wave on electrocardio-

Table 6. Comparison of the 2-dimensional strain analysis between the patients with myocardial bridge (MB) and the controls at the parasternal short axis views (PSX)

PSX	Variables	Control (n=20)	MB (n=18)	p
Septal segment	MV (RS)	48.3±9.7	38.1±17.8	0.02
	MV (CS)	-20.8±9.6	-18.9±4.3	NS
	PM (RS)	68.3±12.8	39.3±11.2	<0.01
	PM (CS)	-25.7±4.3	-26.8±3.2	NS
	Apex (RS)	44.6±15.8	45.4±17.3	NS
	Apex (CS)	-18.8±7.2	-19.8±5.9	NS
Anterior segment	MV (RS)	54.3±13.8	44.3±15.8	0.03
	MV (CS)	-27.2±6.4	-20.6±4.1	NS
	PM (RS)	75.8±15.9	43.6±19.1	<0.01
	PM (CS)	-23.2±4.8	-24.9±2.4	NS
	Apex (RS)	42.2±20.8	43.9±19.9	NS
	Apex (CS)	-18.4±8.7	-19.2±3.1	NS
Lateral segment	MV (RS)	56.9±5.3	55.6±12.8*†	NS
	MV (CS)	-27.2±6.4	-23.5±6.6	NS
	PM (RS)	78.5±16.2	75.1±15.9*†	NS
	PM (CS)	-23.2±4.8	-21.4±3.5	NS
	Apex (RS)	33.2±21.8	34.6±18.6	NS
	Apex (CS)	-17.4±4.7	-18.5±4.5	NS
Posterior segment	MV (RS)	54.7±16.2	55.2±14.7*†	NS

Values are means±SD. *: p<0.05 vs. septal segment, †: p<0.05 vs. anterior segment. MV: mitral valve level, PM: papillary muscle level, RS: radial strain. CS: circumferential strain

graphy to the transition from regional systole to early diastolic lengthening (Tr) was significantly delayed in the MB subjects more than that of the controls (497±20.4 ms vs. 348±12.5 ms, p<0.05) (Fig. 5). The interobserver and intraobserver variability was tested by conducting an independent analysis by two independent observers and by repeated measurement of these segments at another occasion by the same observer. The interobserver variability was less than 20%, and the intraobserver variability was 12%. The main reason for the interobserver variability was the different location of the sample volume. Once the sample volume was placed on a mutually agreed location within the myocardium, the measurements became virtually identical.

Table 7. Segmental difference between the anterior segments and posterior segments in the patients with myocardial bridge (MB) at the papillary muscle level

	Variables	Control (n=20)	MB (n=18)	p
Anterior segments	Radial strain (%)	48.7±12.6	30.9±13.8*	0.01
	Radial displacement (mm)	6.1±1.7	4.8±0.9	0.03
Posterior segments	Radial strain (%)	52.3±15.8	51.8±17.3	NS
	Radial displacement (mm)	5.7±2.9	5.9±1.5	NS
Plateau pattern	Radial strain (%)	0 (0%)	7 (39%)	0.01
	Radial displacement (mm)	0 (0%)	6 (33%)	0.01
Biphasic pattern	Radial strain (%)	1 (5%)	9 (50%)	<0.01
	Radial displacement (mm)	1 (5%)	10 (56%)	<0.01

Values are means±SD. *: p<0.05 vs. posterior segments

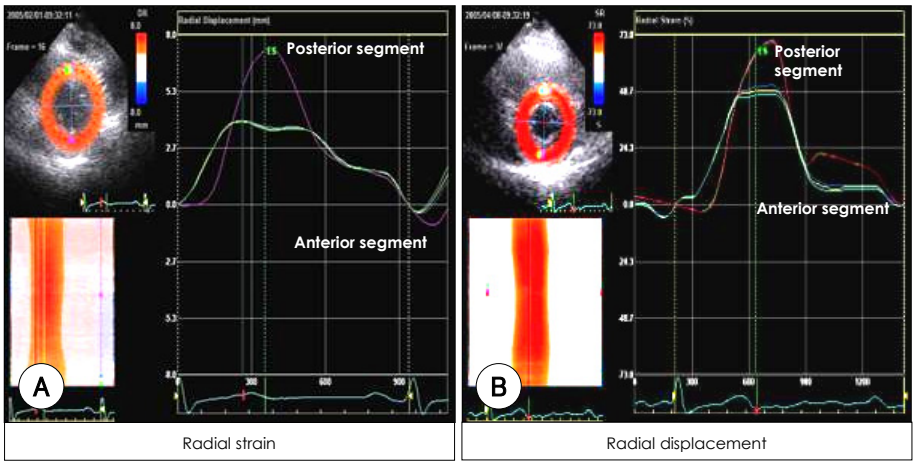

Fig. 4. 2D strain images obtained from a patient with myocardial bridge. Parasternal short axis view. Radial strain (A) and displacement (B) from the papillary muscle level show a plateau pattern at the anterior segments: the peak strain and displacement of the anterior segments were lower than those of the posterior segments. 2D: 2-dimensional.

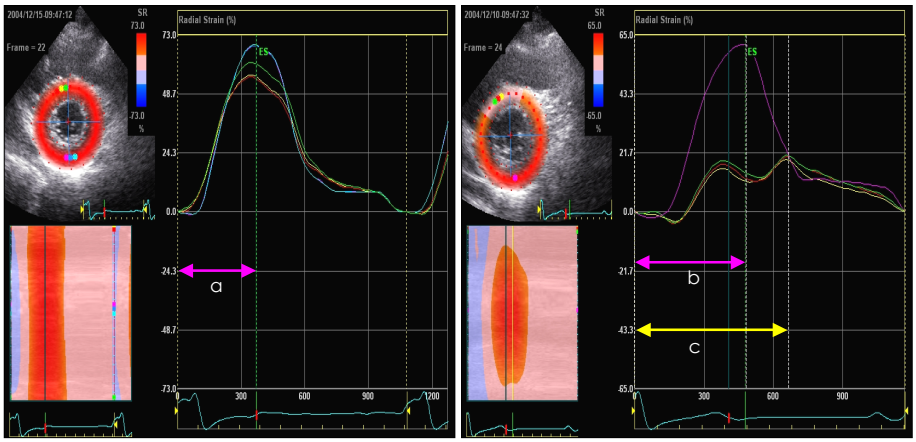

Fig. 5. Comparison of the time from the R wave on electrocardiography to transition from regional systole to early diastolic lengthening (Tr). a: Tr in the controls that is constant throughout the myocardium. b: Tr of the inferior segments of the patients with myocardial bridge. c: Tr of the anterior segments of MB, which is significantly more delayed than in a and b. MB: myocardial bridge.

Discussion

Myocardial bridging occurs when a band of cardiac muscle overlies an intramural segment of a coronary artery, and the intramural segment is referred to as a "tunneled" artery.[1)2)] Until recently, visual interpretation of a coronary angiogram would only reveal the milking effect or the systolic narrowing induced by significant myocardial bridging of a coronary artery. However, it is well recognized that coronary flow predominantly occurs during the diastolic phase of the cardiac cycle. Therefore, it appeared unlikely that this systolic phenomenon could by itself result in myocardial ischemia. Due to compression of the vascular lumen during systole, there is decreased or zero antegrade flow, and at early diastole, release of the resistance in vessels by intraventricular 'throttling' leads to a reduction of resistance, as we observed in normal subjects, and this results in increased antegrade flow. The early diastolic spike is most probably due to the antegrade coronary flow meeting the still compressed (delayed relaxation) and narrowed bridge segment. The subsequent sharp deceleration in the coronary flow velocity results from the release of compression and an increase in the vascular lumen. After the release of the compression, the lumen of the bridge segment remains unchanged in the second half of diastole; therefore, this corresponds to the plateau of the flow pattern at this phase.[10)11)] Although a delay in the diastolic relaxation of the coronary vessel was suspected to occur in myocardial bridge patients, its presence was hard to demonstrate on the coronary angiogram. However, we were better able to understand the hemodynamic disturbances occurring during the diastolic phase of the cardiac cycle in the patients with myocardial bridge by using the new, current era echocardiographic method, i.e., 2-dimensional strain.

The heart, however, has a very complex motion pattern: rotation, contraction and shortening. The current method has a major advantage over the previous methods for automatically detecting wall-motion: the myocardial velocities, strain and strain rate that are obtained from the current non-Doppler-based approach are similar to those measured with using TDI.[12)] During the cardiac cycle, regional deformation of the myocardium occurs in 3 major directions: the longitudinal, circumferential and radial directions. The terms "myocardial strain rate" and "strain" are currently used as the indexes of longitudinal myocardial deformation. The physical definition of strain is the relative change in the length of a material related to its original length. The regional strain rate and strain are derivatives of myocardial velocities. The normal values for LV longitudinal shortening[13)] correspond well with our measurements, and the average peak systolic strain is 19% versus 16.2% as noted in our study. It has previously been shown that the base of the heart descends toward the apex during systole and it reverts during the diastolic phases of early filling and atrial systole. As the moving base descends toward the stationary apex, the tissue velocities have to decrease from the base to the apex.[13)14)] With using strain as determined by TDI, the myocardial displacement determined in our study also showed a base-to-apex gradient. The 2-Dimensional strain method is a tool for quantifying the regional myocardial deformation within a scan plane. Contrary to the strain determined by TDI, the new method is inherently 2D and it is independent of the interrogation angle as it tracks the speckle patterns (the acoustic markers).[15)] Although we did not test the various interrogation angles with respect to the region of interest and the placement of the crystals, we could measure the radial strain and displacement on the parasternal short axis view by employing the 2-dimensional strain, and the radial strain and displacement in the LAD territories were reduced in the patients with myocardial bridge. According to previous studies[16)] systolic strain is constant throughout the wall. Myocardial strain is relatively independent of translational motion and the other through-the-plane motion effects, and it should be relatively uniform throughout the normal LV myocardium. With using high frame rate imaging, the systolic strain measured with the new software was quite uniform throughout the LV. As opposed to normal hearts, the LV of a myocardial bridge was characterized by the marked non-uniformity of myocardial systolic strain. In the longitudinal direction, strain Doppler imaging demonstrated reduced shortening or stretching in the LAD territories. This was in contrast to the nonischemic region in which near-normal shortening was observed. In the second part of our study, making direct comparison with the TDI measurements showed similar results for both the normal patients and the myocardial bridge patients. Determination of the myocardial velocities, the strain rate and strain became possible with the development of TDI.[14-19)] TDI has obvious advantages and it has been accurate when single-dimension motion is being investigated.

Systolic strain showed the best correlation with conventional wall-motion analysis, although the overall correlation was rather modest. The time to the onset of regional relaxation has been proposed as a parameter of the regional myocardial function; this was defined as the time from the R wave on the electrocardiographic tracing to the transition point from contraction to relaxation.[20)] In the healthy volunteers, it was shorter in the mid-segments compared with the apical segments (344 vs. 360 msec, respectively, $p<0.001$). In present study, prolongation of the time to the onset of regional relaxation in the patients with myocardial bridge suggests that myocardial bridge-induced ischemia occurred. Post-systolic compression, defined as the myocardial compression occurring after the end of the LV ejection period, has been sugges-

ted to be an indicator of ischemia and also a marker for the recovery of function after reperfusion. In the present study, the patients with myocardial bridge showed sustained myocardial compression in the LAD territories after the end of the LV ejection period, and this created the plateau or biphasic phenomenon. In this study, we have found for the first time a specific sign of peak strain (the biphasic or plateau phenomenon) surrounding the bridge segment. However, the biphasic or plateau phenomenon was absent in 13% of the patients. This absence might be because compression of the bridging segment was not severe enough to induce a hemodynamic disorder that would lead to the formation of the biphasic phenomenon. The presence of the echocardiographic biphasic or plateau phenomenon doesn't seem to be highly specific for the existence of myocardial bridging, as it can also be observed in normal patients.

Limitations

Tracking of the region of interest was still suboptimal in some patients with hyperdynamic LV function and poor echocardiographic image. It still took from 5 to 10 minutes to achieve satisfactory tracking, and it could not be done in the real-time analysis.

All the patients with myocardial bridge in our study had symptoms compatible to ischemia, so we could not compare the symptomatic myocardial bridge patients with the asymptomatic myocardial bridge patients. Only the parameters of myocardial deformation at rest were investigated, so there could be debate about the presence of resting ischemia in the patients with myocardial bridge. We hope that further investigations on the effects of alterations in the inotropic and chronotropic states that are induced by physical exercise or dobutamine stress echocardiography and the intravenous beta-blockade on the 2-D strain-derived variables in patients with myocardial bridge will allow the determination of more accurate results.

Conclusions

A novel non-Doppler-based software for performing quantitative echocardiography allows automatic wall-motion analysis and the simultaneous measurement of the following parameters: myocardial displacement, strain and the strain rate. These measurements correlated well with those obtained with using TDI in both the normal subjects and the subjects with myocardial bridges. The reduced systolic contraction and delayed diastolic relaxation at the anterior segments of the myocardium seem to be important mechanisms that contribute to the observed ischemia in the symptomatic patients with myocardial bridge. Providing that the image quality is adequate, 2-dimensional strain might have the potential to improve the identification and functional quantification of myocardial bridge.

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