Ultrasonic Tissue Characterization Predicts Myocardial Viability in Early Stage of Reperfused Acute Myocardial Infarction

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Background—The aim of the present study was to characterize temporal changes in cyclic variation of ultrasonic integrated backscatter (IBS), which reflects intrinsic contractile performance, in patients with reperfused acute myocardial infarction (AMI) and to elucidate the clinical value of tissue characterization in predicting myocardial viability.

Methods and Results—We recorded short-axis IBS images before and 3, 7, and 21 days after reperfusion in 26 patients with AMI and obtained the cyclic variation of IBS in the normal and infarct zones. When cyclic variation showed synchrony and asynchrony, we expressed its magnitude as positive and negative values, respectively, called the phase-corrected magnitude. We also measured average wall motion score (dyskinesis, 4; normal, 0) of the infarct segments. The phase-corrected magnitude was lower in the infarct zone than in the normal zone before reperfusion (0.3 $\pm$ 2.5 versus 5.2 $\pm$ 1.7 dB, \( P < .05 \)). At day 3, the phase-corrected magnitude increased by 2.1 $\pm$ 2.6 dB despite no improvement in wall motion. Improvement in wall motion was observed only at day 21. The patients with the phase-corrected magnitude of $\geq 2.0$ dB at day 3 showed significantly lower wall motion score at day 21 than did the other patients (1.7 $\pm$ 0.6 versus 2.4 $\pm$ 0.5, \( P < .01 \)).

Conclusions—In patients with AMI, cyclic variation of IBS is blunted during ischemia but recovers much faster after reperfusion than the improvement in wall motion. The greater phase-corrected magnitude at day 3 may be a predictor of better functional improvement. (Circulation. 1998;97:356-362.)

Key Words: myocardial infarction • echocardiography • reperfusion • myocardial contraction • ultrasonics

One of major problems facing modern cardiology involves the evaluation of myocardial viability within a region of acute ischemic injury. This issue is particularly important in the era of rapid interventional treatment for AMI. Methods based on wall motion analysis fail to differentiate viable tissue from irreversibly damaged myocardium until and unless wall motion improves.1–4 Assessment of regional myocardial perfusion with myocardial contrast echocardiography or assessment of wall motion response to dobutamine stress is another promising approach for evaluating myocardial viability.5–7 However, these approaches are performed in only a small number of acute patients because of technical limitations.

Ultrasonic tissue characterization with IBS offers a promising method for the assessment of myocardial contractile performance independent of wall motion.8–17 Normal myocardium exhibits cardiac cycle-dependent variation of IBS that reflects the intramural contractile performance. In animals, the cyclic variation of IBS is blunted promptly by ischemia and is augmented after reperfusion.18–22 The recovery of wall motion lags behind the recovery of the cyclic variation after brief periods of myocardial ischemia. In patients with reperfused AMI, Milunski et al23 documented that the obvious recovery of cyclic variation of IBS is observed a mean of 7 days after coronary reperfusion regardless of minimal improvement in wall motion. Therefore, the analysis of cyclic variation of IBS may have a potential to detect the viable myocardium early after reperfusion.

In this study, we characterized the temporal changes in cyclic variation of IBS at days 1, 3, 7, and 21 of reperfusion in patients with AMI. We compared these changes with the temporal recovery of regional wall motion to elucidate whether we can predict the functional recovery in individual patient with ultrasonic tissue characterization in the early stage of AMI.

Methods

Study Population

The study group included 26 consecutive patients with AMI who underwent successful coronary angioplasty within 24 hours after symptom onset. Eligible criteria were (1) no prior myocardial infarction, (2) totally or subtotally occluded infarct-related artery (Thrombolysis in Myocardial Infarction grade 0 or 1) at initial coronary angiography, (3) the postprocedural stenosis of <50%, (4) no ischemic

Received July 10, 1997; revision received September 29, 1997; accepted September 30, 1997.

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event during follow-up, (5) patent infarct-related artery 1 month later, and (6) adequate echocardiographic image quality. The diagnosis of AMI was made on the basis of ischemic pain of ≥30 minutes’ duration, ST-segment elevation of ≥1 mm in two or more contiguous ECG leads, and an increase in creatine kinase of more than three times the normal value. β-Blockers and calcium channel blockers were administered at the discretion of the attending physician. No patients were being treated with the positive inotropic agents. One of the investigators obtained informed consent from each patient. The study protocol was approved by the hospital ethics committee.

Acoustic Densitometry
We used a special software package (Acoustic Densitometry) developed by Hewlett-Packard incorporated into a commercially available echocardiograph (SONOS 1500). This system is capable of providing either conventional echocardiographic images or two-dimensional images in which gray level is displayed proportional to IBS amplitude. Sixty frames from consecutive cardiac cycles (30 frames/s) are displayed after scan conversion and are stored on the optical disk. The system has a unique feature by which the transmit power, log compression, and time-gain compensation values are displayed on the screen and are stored with the images, which allows an operator to adjust the system to the same values at any follow-up examination. The dynamic range of the IBS processor is 40 dB.

Data Analysis
We analyzed the digitally acquired images with the acoustic densitometry package to construct time-intensity waveforms of the IBS. We divided the short-axis image of the left ventricle into eight segments. We defined the segment at risk as the segment showing severe hypokinesia, akinnesia, or dyskinesia at baseline study. In the analysis, we excluded the segments of the inferior septum and the lateral wall because of the unreliability of waveform of the cyclic variation in these segments. In these segments, the angle between ultrasound beam and fiber orientation is shallow, so the magnitude of IBS is significantly reduced.24,25 We placed the ovoid region of interest at the center of the segments at risk and in the remote normal region in each patient at each examination. We used the largest possible region of interest, which did not include endocardial and epicardial reflectors. An experienced echocardiographer manually adjusted the location of the site on a frame-by-frame basis to keep the site within the myocardial midwall throughout a cardiac cycle. Then, a curve of IBS versus time was reconstructed. We determined the magnitude (in decibels) of cyclic variation of IBS as the difference between the minimal and maximal values in a cardiac cycle averaged over at least two consecutive beats. Absolute calibration is unnecessary for measurement of the magnitude of cyclic variation. Because the regional contraction in the infarct zone may not be necessarily synchronized exactly with global contractile events, we calculated a time delay for regional cyclic variation with respect to global ventricular mechanical systole.26,27 After measuring the interval from the upstroke of QRS complex to the nadir of the cyclic variation at each site, we divided the value by QT interval to determine the normalized delay (unlabeled). We also corrected the magnitude of cyclic variation with respect to the phase of regional contraction. In this study, a mean of the normalized delay in the normal segment was 1.0 ± 0.1. Therefore, if the normalized delay value was >1.2 (mean +2 SD of normal), we considered it indicative of asynchronous (or delayed) contraction or passive stretching and multiplied the magnitude by −1.0 (the phase-corrected magnitude). This is an approximation to the phase-weighted amplitude.19,20 If the normalized delay value is ≥1.2, the phase-corrected magnitude is the same as the measured magnitude value.

Two independent observers who blinded to patients’ clinical data analyzed the wall motion at the site of region of interest in each study with following scoring system: 4 indicates dyskinetic; 3, akinetic; 2, severely hypokineti; 1, hypokineti; and 0, normal. In the evaluation, we carefully examined the systolic thickening in the central portion of each segment. In cases of disagreement, a third observer established the consensus. We also evaluated the recovery of wall motion in the risk segments showing asynergy at baseline study. To do this, we divided the left ventricle into 17 segments (8 segments on each short-axis slice at the levels of the mitral valve and midpapillary muscle, and apical segment on the apical long-axis view).28 The same observers scored each segment using the scoring system previously described. We defined wall motion score index as an average of segmental scores of the risk segments at days 1 and 21.

Reproducibility of Data
We determined intraobserver and interobserver variabilities of measuring the magnitude and normalized delay value of cyclic variation of IBS by measuring the two variables in 10 randomly selected records twice by the same observer and by two independent observers, respectively. Intraobserver and interobserver variabilities of the magnitude of IBS were 4.2 ± 4.0% and 5.1 ± 4.2% (absolute difference), respectively. Intraobserver and interobserver variabilities of normalized delay values were 4.2 ± 3.2% and 4.7 ± 2.4% (absolute difference), respectively.

Statistics
All data are expressed as mean ± SD. Multiple comparisons were made with a one-way ANOVA, and individual data were compared with the use of Scheffé’s F test for factor analysis. Statistical analysis of temporal changes in certain variables was computed with ANOVA and Scheffé’s F test for repeated measures. Differences were considered significant at P < .05.

Results
Patient Characteristics
Among the 26 patients (mean age, 59 ± 12 years; age range, 36 to 77 years), 23 patients (81%) were male and 3 patients were female (Table). All patients had one-vessel disease. Anterior, inferior, and posterior myocardial infarction was found in 16, 7, and 3 patients, respectively. The mean time from the symptomatic onset to coronary reperfusion was 6.5 ± 4.6 hours. The peak creatine kinase level was 3052 ± 2050 IU/L. Twenty-three patients (92%) subsequently developed Q-wave infarction, and the other 3 manifested non-Q-wave infarction.

Cyclic Variation of IBS Before Reperfusion
Cyclic variation of IBS was present in all 26 normal regions and averaged 5.2 ± 1.7 dB in magnitude. The average normalized
The time delay of cyclic variation was 0.95±0.19 (Figs 1 and 2). In the short-axis image, the center of all infarct segments showed akinesia at baseline study. The magnitude of cyclic variation in the infarct segments decreased significantly to 1.9±1.7 dB (P<.05). No cyclic variation was detectable in 7 of 26 infarct segments. Even when these 7 regions were excluded from the average, the magnitude of cyclic variation was still lower than normal values, averaging 2.5±1.5 dB. Normalized time delay of cyclic variation was measurable in 16 infarct segments, and the value was significantly longer than that in the normal zone.

**Figure 1.** Cyclic variation of IBS in the normal posterior wall (left) and infarct anterior wall (right) in a patient with anterior wall myocardial infarction. Cyclic variation in the normal posterior wall shows synchronous contraction, but it shows an asynchronous pattern in the infarct anterior wall. The magnitude is significantly lower in the infarct zone than in the normal zone.

**Figure 2.** Normalized time delay (left), magnitude (middle), and phase-corrected magnitude (right) of cyclic variation of IBS at baseline in the normal and infarct zones. Normalized time delay was significantly greater and magnitude and phase-corrected magnitude were significantly lower in the infarct zone than in the normal zone. Values are expressed as mean±SD. *P<.05, †P<.01.

**Clinical Characteristics and IBS Data for 26 Study Patients**

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CV indicates cyclic variation; delay, delay of cyclic variation; A, anterior wall; I, inferior wall; and P, posterior wall.
(1.25±0.49, P<.05). Normalized delay value was >1.2 in 7 patients, indicating asynchronous contraction or passive stretching. Thus, the phase-corrected magnitude was significantly lower in the infarct regions than in the normal regions (0.3±2.5 versus 5.2±1.7, P<.05).

**Temporal Changes in Cyclic Variation and Wall Motion After Reperfusion**

Cyclic variation of IBS was present in 23 of 26 infarct segments at 3 days later (Figs 3 and 4). Cyclic variation was detectable at this phase in 7 patients in whom no cyclic variation was detectable at baseline. The number of patients showing asynchronous contraction decreased from 7 to 3 at day 3 (Table). As a whole, there was no significant changes in the magnitude of cyclic variation in the infarct region from baseline to the second study, and it was still lower than that in the remote normal region (2.6±2.0 versus 5.2±1.4 dB, P<.05). The normalized time delay value slightly decreased to 1.10±0.38 at day 3, but it was still higher than that in the normal zone (1.10±0.38 versus 0.93±0.15, P<.05). Phase-corrected magnitude of cyclic variation significantly increased from baseline to day 3 (0.3±2.5 versus 2.1±2.6 dB, P<.05) (Fig 4). However, there was no significant improvement in wall motion in the center of the infarct segment. The phase-corrected magnitude increased to 3.0±3.6 at day 7, but it did not show a significant increase after that. In contrast, wall motion score significantly decreased only at day 21 (day 1 versus day 21, 3.0±0.0 versus 2.4±0.8, P<.05).

**Prediction of Wall Motion Improvement From Phase-Corrected Magnitude**

We assessed the potential of the phase-corrected magnitude for predicting functional improvement in the infarct segments (Figs 5 and 6). Using an ROC curve, we examined the sensitivity and specificity of various cutoff points of phase-corrected magnitude at day 3 for predicting viable myocardium. The ROC curve is a plot of sensitivity against 1−specificity as the positive/negative cutoff point is varied. Definition of viable myocardium was wall motion score index of ≥2, in the convalescent stage. Based on this curve configuration, we considered the optimal cutoff point to predict viable myocardium appears to lie around the phase-corrected magnitude of 2 dB, which is a median value of this magnitude at day 3.

The patients were divided into two groups according to this value for phase-corrected magnitude: those with the phase-corrected magnitude of ≥2.0 dB were considered group A (14 patients), and those with the phase-corrected magnitude of
Discussion

Before coronary reperfusion, the cyclic variation of IBS in the infarct zone is markedly blunted and even shows asynchronous waveform in patients with AMI. The phase-corrected magnitude of cyclic variation significantly increased despite the minimal improvement in wall motion 3 days after reperfusion. It progressively increased until day 7. An improvement in wall motion, however, was found only at day 21; therefore, an increase in the phase-corrected magnitude precedes an improvement of wall motion. In addition, our data indicated that the greater values for the phase-corrected magnitude at day 3 are associated with the better functional improvement of the postischemic myocardium. Thus, the ultrasonic tissue characterization can provide a useful measure of intramural contractile function, relatively independently of wall motion, and may permit the prediction of wall motion recovery in very early stage of reperfused AMI.

Cyclic Variation of IBS in Infarct Segment

Several experimental and clinical studies have documented that the cyclic variation of IBS is promptly blunted and often shows substantial time delay compared with left ventricular systole, which implies asynchrony, during myocardial ischemia. In patients with AMI, we also observed the marked reduction in the magnitude of cyclic variation and the significant increase in the time delay in the infarct segment before reperfusion. The pattern of cyclic variation, however, varies among patients. Cyclic variation was not detectable in 7 of 26 patients. Apparent asynchronous contraction was observed in 7 patients (27%). In 5 patients (patients 1, 7, 23, 25, and 26), the absolute magnitude of cyclic variation was comparable to or greater than that in the normal segment, but their pattern appears to be a mirror image of normal cyclic variation pattern. This asynchronous pattern of cyclic variation may be caused by stretching or passive distention of ischemic myocardium during systole. Although wall motion of the infarct zone was evaluated as akinetic before reperfusion, the intramyocardial contraction pattern varies among patients and the analysis of IBS is useful to assess the pathophysiology of the wall motion abnormalities.

Several mechanisms are postulated to explain the cyclic variation of IBS: (1) changes in acoustic impedance, which is related to passive elastance; (2) changes in fiber orientation or shape from diastole to systole; and (3) changes in elastic modules during sarcomere shortening. In open-chest dogs, Wickline et al.22 showed that cyclic variation of IBS in the myocardium parallels contractile performance. However, wall thickening is not linearly related to the magnitude of the cyclic variation in dogs with acute coronary occlusion followed by reperfusion.19,30 Although influenced by wall thickening, cyclic variation of IBS appears to reflect the intramural contractile performance rather than geometric phenomenon.6–10

Mechanisms responsible for the temporal delay of cyclic variation of IBS are also unclear. In open-chest dogs, Brown et al.31 documented postsystolic shortening and thickening of myocardium during coronary ligation. Early diastolic or post-systolic events may be related to persisting contraction or delayed relaxation within the ischemic myocardium2,23 and perhaps to effects of stretching or passive distention,11 although it is hard to differentiate these events. Thus, the delayed cyclic variation that we observed may reflect postrystolic events and ultimately provide an approach for their detection and quantification. Because these events are considered to be unfavorable to global left ventricular contraction, we expressed the corrected magnitude of cyclic variation as a negative value if the cyclic variation exhibited asynchronous contraction.

Effect of Reperfusion

In animals, the reduced magnitude of cyclic variation of IBS during ischemia gradually increased after coronary reperfusion.18–22 Milusnski et al.23 documented that partial recovery of cyclic variation, which included an increase in the magnitude of cyclic variation and a decrease in the normalized time delay, was observed at a mean of 7 days after reperfusion in patients with AMI despite minimal recovery of wall motion. In this study, we investigated the temporal changes in the cyclic variation 3 days after reperfusion. The results are a little different from their results because temporal changes in cyclic variation varied among patients with the pattern of cyclic variation of IBS at baseline. An increase in the magnitude of cyclic variation was observed 3 days after reperfusion in 15 of 19 patients showing synchronous or no detectable cyclic variation at baseline study, but the other 4 patients showed a decrease in the magnitude. In these 4 patients, the contribution of progressive myocardial damage after reperfusion was speculative. The patients showing the asynchronous pattern of cyclic variation at baseline represented a decrease in the absolute magnitude, and thus coronary reperfusion does not always lead to a increase in the phase-corrected magnitude of cyclic variation. In the majority of these patients, however, the normalized delay decreased to the normal range, and the corrected magnitude increased 3 days after reperfusion. These data imply that reperfusion usually augments the cyclic variation in case of synchronous contraction and diminishes the delayed relaxation or passive stretch in cases of asynchronous segmental contraction, resulting in an increase in the corrected magnitude as early as 3 days after reperfusion. At this phase, however, no substantial improvement in the segmental motion was detectable in the center of myocardial infarction.

Milusnski et al.23 observed the temporal changes in wall motion and cyclic variation of IBS in the infarct zone until a mean of 7 days after reperfusion. However, the temporal changes in the phase-corrected magnitude of the cyclic variation until the convalescent stage of AMI and their relation to the recovery of wall motion remain unknown. Our data demonstrated that the phase-corrected magnitude increased.
Despite achievement of coronary recanalization in the early stage of AMI and because the amount of viable myocardium determines the final functional outcomes and patients’ prognosis.

**Study Limitations**

Cyclic variation of IBS is dependent on the angle between fiber orientation and ultrasonic beam, called anisotropy. Time delay also varies systematically with fiber orientation. Data in this study were obtained exclusively from the anteroseptum and inferior and posterior regions in the parasternal short-axis views, in which myocardial fiber of these segments is oriented nearly perpendicular to the ultrasound beam, thereby avoiding problems with anisotropy as much as possible. In addition, the analysis is largely dependent on the image quality.

One of the disadvantages of IBS imaging in its current form is that there is a relatively narrow dynamic range before which system saturation occurs. However, the problem regarding narrow dynamic range can be minimized by reducing the transmit power. Another disadvantage of IBS imaging in its current form is a significant degradation in spatial resolution compared with conventional two-dimensional echocardiography.

The IBS signal is influenced by gain setting. We set the time-gain control to the identical value in each patient, and only total gain was controlled to clearly depict the IBS image. The difference in total gain should not influence the magnitude of the cyclic variation of IBS.

**Clinical Implications**

Detection of regional wall motion abnormalities through conventional two-dimensional echocardiography facilitates the localization of the injured or infarct segments. Its use, however, may not delineate the overall extent of myocardial salvage early after reperfusion until and unless the recovery of wall motion has occurred. Our results documented that an increase in the phase-corrected magnitude precedes the improvement in wall motion in patients with reperfused AMI. Such information cannot be obtained through qualitative or quantitative analysis of wall motion alone. Clinically, the analysis of the cyclic variation of IBS may allow the rapid assessment of regional myocardial viability despite no or minimal improvement in wall motion.

Myocardial contrast echocardiography or dobutamine stress echocardiography is another method by which to assess myocardial viability in the early stage of AMI. Because of technical limitations, these methods cannot be applied to all acute patients. Compared with these methods, ultrasonic tissue characterization is simple; therefore, ultrasonic tissue characterization should be a new diagnostic modality for evaluating myocardial viability at the bedside in patients with AMI.

**Acknowledgments**

The authors greatly acknowledge the excellent technical assistance of Yuzo Sakagami, Masakazu Ueda, Naoki Jonishi, and Hideshi Shimokawa.

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Tissue Characterization in AMI


Ultrasonic Tissue Characterization Predicts Myocardial Viability in Early Stage of Reperfused Acute Myocardial Infarction
Shin Takiuchi, Hiroshi Ito, Katsuomi Iwakura, Yoshiaki Taniyama, Nagahiro Nishikawa, Tohru Masuyama, Masatsugu Hori, Yorihiko Higashino, Kenshi Fujii and Takazo Minamino

Circulation. 1998;97:356-362
doi: 10.1161/01.CIR.97.4.356

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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