Magnetic Resonance in Coronary and Valve Disease

Relationship of Contractile Function to Transmural Extent of Infarction in Patients With Chronic Coronary Artery Disease

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OBJECTIVES
We sought to determine the relationship of contractile function to the transmural extent of infarction (TEI) in patients with chronic coronary artery disease.

BACKGROUND
In the setting of reperfused, chronic myocardial infarction (MI), the relationship of contractile function to the TEI has not been established.

METHODS
We studied function by cine magnetic resonance imaging (MRI) and the TEI by contrast-enhanced MRI in 31 patients with single-vessel disease 162 ± 62 days after reperfused first MI.

RESULTS
Of all 516 segments with MI, blinded observers were unable to detect abnormal thickening in 193 (37%), and wall thickening measured quantitatively in these segments was 66 ± 28%.

Of the 193 segments, 163 (84%) were infarcts limited to the subendocardium. The average TEI reached 53% before half of the patients had abnormal contractile function. When patients with small MI (<5% of total left ventricular [LV] mass) were excluded, the average TEI reached 43% before half the patients had abnormal function. In subjects with small MI (<5% of total LV mass [n = 13]), even segments with TEI >75% had normal function (14 of 14) because they were surrounded by normally moving neighbor segments.

CONCLUSIONS
In the setting of reperfused chronic MI, the TEI approaches 50% before contractile dysfunction can be systematically identified. Contractile function cannot be used to rule out chronic MI.

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The diagnosis of myocardial infarction (MI) is of fundamental clinical importance. While MI is often associated with impaired contractile function, the relationship between infarction and contractile function is complex. For example, Lieberman et al. (1) demonstrated that sizing of myocardial infarcts based on contractile function results in an overestimation of infarct size (2–5) because of a “threshold phenomenon” caused by a “disproportionate role of the subendocardial layer of the myocardium in overall wall thickening” (WT) (6,7). Accordingly, the spatial extent of contractile dysfunction may be larger than the spatial extent of the infarct itself. It is important to recognize, however, that many of the studies supporting the concept of infarct overestimation were performed before recognizing that ischemic but viable tissue surrounding the infarct can exhibit contractile dysfunction for several days after reperfusion (“myocardial stunning” [4,7–9]), complicating interpretation of existing data.

In patients in whom successful revascularization has been achieved contractile function systematically improves in the weeks and months that follow (8–12). This improvement underscores that data acquired in the acute setting, where effects such as “stunning” may play a role, do not apply to the chronic setting. When literature reports of contractile function in patients with infarction are divided into acute (2,4–7) and chronic, it becomes apparent that the relationship of WT to transmural extent of infarction (TEI) has not been established in patients with chronic MI.

Contrast-enhanced magnetic resonance imaging (ceMRI) can be used to examine the TEI in patients with chronic MI (13,14). In the present study, we used ceMRI to examine the TEI and cine magnetic resonance imaging (MRI) to examine contractile function in the same imaging session to eliminate errors due to misregistration. The magnetic resonance (MR) images were acquired five months post-infarct in patients with MI defined by cardiac enzymes for whom cardiac catheterization showed both single-vessel disease and successful reperfusion. The data underscore that the relationship of contractile function to the TEI in this chronic setting is fundamentally different from that of an acute setting reported by other investigators (1,15,16).
METHODS

Patient population. This study was approved by the Institutional Review Board of Northwestern University, and all patients gave informed consent. Thirty-one patients (27 men and 4 women) admitted to the coronary care units at Northwestern Memorial Hospital (20 men and 4 women) and Veterans Administration Lakeside Hospital (7 men), presenting with first MI defined by cardiac enzymes (creatinine kinase-MB fraction >9 μg/ml), were sequentially enrolled. Patients were included if they had: 1) no previous history of MI; 2) single-vessel disease defined by cardiac catheterization (no additional stenosis >50%); and 3) underwent successful primary angioplasty and had no contraindications to MRI. All patients were scanned approximately five months after reperfused first MI (162 ± 62 days). Twelve patients were also scanned acutely as part of another study (12). No patient was excluded from the study for technical or image quality reasons, and none of the patients had clinical evidence of recurrent ischemia between revascularization and MRI. Table 1 summarizes the clinical characteristics of the patient population.

MRI protocol. The MRI was electrocardiogram-gated, and all images were acquired during repeated breath-holds. Both cine and contrast-enhanced short-axis (SAX) MR images were prescribed every 10 mm (slice thickness 5 to 6 mm) from base to apex (6 to 7 cine slices and 6 to 7 corresponding contrast-enhanced slices per heart). In-plane image resolution was typically 1.4 × 1.8 mm. Cine MRI was performed using a steady-state free precession sequence, and ceMRI images were acquired using an inversion recovery gradient echo pulse sequence described in detail elsewhere.

Abbreviations and Acronyms

- ceMRI = contrast-enhanced magnetic resonance imaging
- LAX = long axis
- LV = left ventricle/ventricular
- MI = myocardial infarction
- MR = magnetic resonance
- MRI = magnetic resonance imaging
- SAX = short axis
- TEI = transmural extent of infarction
- WM = wall motion
- WT = wall thickening

Table 1. Patient Characteristics

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All 59 ± 11 N/A 2.3 ± 0.3 58 ± 16 N/A 1,462 ± 446 152 ± 97 12 ± 5 162 ± 62

Values in “All” row are expressed as mean ± SD.

CK = creatine kinase; CK-MB = creatine kinase-MB fraction; EF = ejection fraction; IRA = infarct-related artery; LAD = left anterior descending; LCX = left circumflex artery; %LV = percent of left ventricular mass; MRI = magnetic resonance imaging; N/A = non-applicable; RCA = right coronary artery.
where (17). The contrast agent (gadoteridol, Bracco Diagnostics Inc., Princeton, New Jersey) dose was 0.15 mmol/kg.

**Segmental model.** All images were analyzed using a 72-segment model: 6 SAX slices × 12 segments per slice (11). To address issues concerning misregistration and through-plane motion, we additionally analyzed the quantitative patient data using the American Heart Association/American College of Cardiology (AHA/ACC) recommended 17-segment model (18) and, separately, long-axis (LAX) images alone using six segments.

**Wall thickening.** Cine images were analyzed both qualitatively and quantitatively. For qualitative analysis, patient images were randomized with images from eight normal volunteers and interpreted by two observers. The observers were blinded to the subjects’ identity, the presence or absence of MI, and the contrast enhancement. Segmental WT was scored by consensus according to the following scheme: 0 = normal, 1 = mild-to-moderate hypokinesis, 2 = severe hypokinesis, 3 = akinesis, and 4 = dyskinesis.

For the quantitative analysis, endocardial and epicardial contours were outlined manually. Based on these contours, WT was determined by computer using the centerline method (19). Neighboring segments were defined as segments adjacent to the index segment in the same plane (left and right) or one plane above or below the index segment (basal and apical).

**TEI.** The TEI was determined quantitatively based on contrast-enhanced images by observers blinded to patient identity and the cine images. For each segment, the total area and, separately, the hyperenhanced area were outlined. Hyperenhanced pixels were defined as those with image intensities more than two standard deviations above the mean of image intensities in a remote myocardial region in the same image. The TEI was calculated as: TEI (%) = HA · 100/(TA), where HA = hyperenhanced area and TA = total area. The average TEI on a per patient basis was defined as the average TEI of all segments with TEI > 0%.

**Statistics.** Continuous data are expressed as mean ± SD. Linear regression analysis was used to assess the relationships between segmental wall motion (WM) score or thickening and the extent of hyperenhancement as well as WM and hyperenhancement of neighboring segments. The linear regression analysis was performed using mixed effects models (SPLUS 2000, Mathsoft, Seattle, Washington) because of the nonindependence and varying number of segments for each subject. The final model results are based upon the combined estimates from all subjects after adjusting for the number of segments from each subject. All statistical tests were two-tailed, and p < 0.05 was regarded as significant.

**RESULTS**

**Segmental analysis.** In our patient population, 328 (15%) of the 2,232 segments (31 patients × 72 segments = 2,232) were either within the left ventricular (LV) outflow tract or located apically to be well visualized by the MR approach used in the present study, yielding 1,904 segments for analysis. Interobserver (2 ± 0.5%) and intraobserver (2 ± 0.5%) variability were not statistically significant (p = NS).

**Relationship of contractile function to TEI.** Figure 1 shows a typical result comparing contractile function with the TEI in a patient. In this example, significant WT was observed despite the presence of non-transmural infarction (arrows in Fig. 1). In the blinded analysis, WT was judged to be normal. Quantitative WT in the region of the infarct was not statistically different from remote regions (p = NS).

Figure 2 displays the results for all patients qualitatively (Fig. 2A) and quantitatively (Fig. 2B). Of the 160 segments with TEI of 1% to 25% (Fig. 2A), blinded observers were
to detect abnormal WT in 120 (75%). Of the 516 segments with any infarction (Fig. 2B), 516 = 160 + 168 + 86 + 102, blinded observers were unable to detect abnormal WT in 193 (37%), and WT measured quantitatively in these segments was 66 ± 28%. Of these 193 segments, 163 (84%) were subendocardial infarcts with a TEI of 50% or less. End-diastolic wall thickness in infarcted segments with normal WM was not statistically different compared with non-infarcted segments (6.6 ± 1.5 mm vs. 6.0 ± 1.8 mm, p = NS).

To evaluate the potential role of misregistration between cine and contrast-enhanced images using the 72-segment model, the data were reanalyzed using the ACC-recommended 17-segment model (Fig. 3A). To evaluate the potential role of through-plane motion, LAX images were analyzed without the SAX images using a six-segment model (Fig. 3B). Results of these two additional analysis methods (Fig. 3) were similar to those of the 72-segment model (Fig. 2).

Figure 4 summarizes the results by patient as opposed to by segment. In Figure 4, the cumulative percent of patients with abnormal contraction is plotted against the average TEI. The cumulative percent with abnormal contraction was defined as all subjects (31 patients) minus those with completely normal contraction (WM score = 0 for all segments) divided by the number of subjects. The average TEI was defined as the sum of TEI for all segments with hyperenhancement divided by the total number of segments with hyperenhancement. Of the 31 patients with healed MI, 10 had normal function and therefore the curve ends at 68% (100% · [31 – 10]/31). For all MIs, the TEI reached 53% before half of the patients had abnormal function (right-hand dotted line in Fig. 4). When the 13 patients with small infarcts (≤5% LV, Table 1) were excluded (filled triangles in Fig. 4), 2 of the 18 remaining patients had normal function and the curve ends at 88% (100% · [18 – 2]/18). For patients with MI >5% LV, the TEI reached 43% before one-half of the patients had abnormal function (left-hand dotted line in Fig. 4).

The neighbor effect. Figure 5 shows a typical result of contractile function in regions with nearly transmural infarction surrounded by normal moving neighbors. In this example, significant WT was observed despite the presence of nearly transmural infarction (arrows in Figure 5). In the blinded analysis of these images, WT was judged to be normal. Quantitative WT and thickness in the region of the infarct were not different from that in remote regions (p = NS).

Figure 6 depicts function based on whether neighboring segments exhibited abnormal or normal contractile function both qualitatively (Fig. 6A) and quantitatively (Fig. 6B). For the qualitative data, a segment with abnormal neighbors was defined as any segment with three or more neighbors contracting abnormally (WM score not equal 0). For the quantitative data, a segment with abnormal neighbors was defined as any segment with three or more neighbors found to be thickening <30% by computer analysis. As seen in Figure 6, contractile function of any given segment was statistically different depending on whether or not neighboring segments were contracting normally (p > 0.05). For subjects with infarct size ≤5% of total LV mass (n = 13), 14 of 14 (100%) segments with TEI > 75% had normal WM if surrounded by normal moving neighbors.

Results of the multivariate analysis of factors that influence contractile function of a given segment are shown in Table 2. The analysis considered three factors: 1) contractile function of neighboring segments (with vs. without three or more abnormal moving neighbors); 2) TEI of neighboring segments (with vs. without three or more neighbors showing HE); and 3) TEI of the index segment. In general, all three factors were statistically significant at the p < 0.05 level, although based on the statistic the factor with the strongest influence on contractile function for a given
segment was the contractile function of neighboring segments (Table 2).

**DISCUSSION**

We found that in the setting of reperfused chronic MI, contractile function often appears normal in regions of non-transmural infarcts (e.g., TEI 1% to 25%, Figs. 2 and 3). Our data also demonstrate that WT can appear to be normal even in regions of nearly transmural infarcts, if the total infarct volume is small (<5% of the LV mass), and the infarct region is surrounded by normal moving neighbor segments. These current findings, obtained in a chronic setting, are fundamentally different from those previously reported by other investigators in the acute setting (1,15), because our data demonstrate that the "threshold phenomenon" observed in an acute and/or non-reperfused setting is not observed in the setting of chronic reperfused infarction.

**Relationship of contractile function to TEI.** Previous studies indicate that sizing of myocardial infarcts based on contractile function (e.g., by echocardiography) results in an overestimation of infarct size (2–5). This overestimation is thought to relate to a "threshold phenomenon" which is known as an abrupt cessation of systolic function, occurring when the TEI exceeds approximately 20%, as described previously (Figs. 2 and 3). Rather, we found that contractile function can appear to be normal for TEI approaching 50%. This apparent discrepancy may be explained by the different settings employed in our study. Previous studies examined contractile function for infarcts that were acute (1–3,20,21) and/or non-reperfused (1,6,7,22), whereas the data of our study specifically address the setting of chronic, reperfused infarction. In the setting of acute infarction contractile function may be reduced secondary to myocardial stunning. In addition, contractile function may be reduced in the setting of non-reperfused infarction due to ongoing ischemia. Myocardial stunning, however, resolves on a time scale of days to weeks and would not be expected to influence contractile function in our study because we scanned patients approximately five months after the infarct. Furthermore, ongoing ischemia would not be expected in our study because we excluded patients without successful reperfusion. Our data demonstrate that the "threshold phenomenon" observed in an acute and/or non-reperfused setting is not observed in the setting of chronic reperfused infarction.

**The neighbor effect.** The data of most previous reports addressing the effects of neighboring myocardial segments on contractile function were acquired in an acute setting (2,3,5,21). Accordingly, the relationship of contractile function to infarct transmurality in acute versus chronic settings is separately discussed in the following paragraphs.

In addition to the "threshold phenomenon" (1), the overestimation of infarct size in the acute setting (2,4,5) may also be explained by an effect known as "tethering," defined as dysfunction of perfused, viable myocardium adjacent to
infarcted or ischemic myocardial segments (2,3,21). Tethering is thought to be present approximately 10 to 15 mm around the border of infarcted or ischemic segments and to impair the movement of healthy neighboring segments owing to mechanical effects on their muscular fibers (3). It is important to recognize, however, that much of the data supporting the concept of tethering was acquired in an acute (2,3) or not reperfused (21) setting.

In the chronic setting, few if any previous reports have systematically described the effects of neighboring myocardial segments on contractile function. In the current study, we found that normally contracting segments surrounding the infarcted area can result in the impression of thickening in infarcted segments. This influence of neighboring segments on contractile function may also be thought of as a form of “inverse tethering.” Importantly, however, the “tethering mechanism” described by previous investigators in the acute setting refers to a reduction in contractile function by non-functioning neighbors, whereas our data suggest that “inverse tethering” can also act in the opposite direction, resulting in the appearance of improved contractile function in infarcted segments due to contraction of neighboring segments (Fig. 5).

Our observation that moving segments surrounding the infarcted area can result in the appearance of thickening in infarcted segments could also be explained by through-plane motion of the heart during cardiac contraction. In this case, the infarcted segment was not actually thickening, but rather, new myocardium had traveled into the imaging plane. To test this, we studied contractile function on a per patient basis and found that 10 of the 31 patients in our study (all of whom had MI) had completely normal WM

**Figure 4.** Summary of the results by patient as opposed to segmentally. See text for details. LV = left ventricular; MI = myocardial infarction; TEI = transmural extent of infarction.

![Figure 4](http://dcmrc.mc.duke.edu/mahrholdt/fig4/)

**Figure 5.** Typical results of contractile function in regions with nearly transmural infarction surrounded by normal moving neighbors in Patient #14. The full cine version of Figure 5 can be viewed at: http://dcmrc.mc.duke.edu/mahrholdt/fig5/.
throughout the entire LV. This observation suggests that through-plane motion may at best play a secondary role. Regardless of the underlying mechanism, however, our data demonstrate that in a routine clinical setting the presence of MI may be difficult to detect when based purely on the observation of contractile function.

Clinical relevance. Previous studies have established that both small and large myocardial infarcts are associated with a poorer prognosis (23–26). Despite the recognition that both small and large infarcts are prognostically important, there is only a limited period of time in which biochemical evidence such as elevated serum enzymes can be used to detect MI. Once serum enzymes have returned to baseline, infarcts can only be detected by the observation of contractile abnormalities, the reduction of wall thickness by echocardiography and/or ventriculography, or by observation of a fixed defect by nuclear scintigraphy.

Many authors report that using contractile function to
assess MI consistently leads to overestimation of infarct size (2–5). Our data demonstrate that this same approach can also systematically underestimate infarct size if contractile function is examined in a reperfused chronic setting. This finding implies that, in the absence of biochemical evidence of infarction and in the absence of significant wall thinning which only occurs in larger transmural infarcts, imaging techniques in which the primary definition of infarction is impaired contractile function may systematically miss patients with subendocardial chronic infarcts. The frequency with which these non-transmural infarcts are missed using current routine clinical approaches is unknown and requires further investigation.

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REFERENCES