Journal of the American College of Cardiology
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Published by Elsevier Science Inc.

Vol. 36, No. 6, 2000
ISSN 0735-1097/00/$20.00
PII S0735-1097(00)00945-1

Contractile Reserve and Contrast Uptake Pattern by Magnetic Resonance Imaging and Functional Recovery After Reperfused Myocardial Infarction

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OBJECTIVES We hypothesized that contrast-enhanced and dobutamine tagged magnetic resonance imaging (MRI) could investigate microvascular integrity and contractile reserve of reperfused myocardial infarction (MI) in one examination.

BACKGROUND In reperfused MI, microvascular integrity and contractile reserve are important determinants of functional recovery.

METHODS Twenty-three patients with a reperfused first MI were studied. On day 3 ± 1 after MI, patients underwent tagged MRI at baseline and during infusion of 5 and 10 μg/kg/min of dobutamine followed by contrast-enhanced MRI (first pass and delayed imaging) after a bolus infusion of gadolinium-diethylenetriaminepenta-acetic acid. Tagged MRI was performed 9 ± 1 weeks later (follow-up). Eighty-four transmural regions with hyperenhancement on delayed contrast-enhanced images were defined as COMB (first pass hypoenhancement) or HYPER (normal first pass signal enhancement). Percent circumferential segment shortening was measured within the subendocardium and subepicardum of each region of HYPER or COMB at baseline, peak dobutamine and follow-up.

RESULTS Shortening improved in COMB regions from 4 ± 1% at baseline to 10 ± 1% at peak dobutamine and 10 ± 1% at follow-up, respectively ($p < 0.0003$ vs. baseline for both). The HYPER regions likewise improved from 10 ± 1% at baseline to 16 ± 1% and 17 ± 1%, respectively ($p < 0.0004$ vs. baseline for both). Function within COMB regions was less than that of HYPER at baseline, peak dobutamine and follow-up ($p < 0.0003$ for all).

CONCLUSIONS Dobutamine magnetic resonance tagging and contrast enhanced MRI are complementary in assessing functional recovery after reperfused MI. Regions of delayed contrast hyperenhancement demonstrate both contractile reserve and late functional recovery. However, if these regions demonstrate first pass contrast hypoenhancement, they are associated with greater myocardial damage. (J Am Coll Cardiol 2000;36:1835–40) © 2000 by the American College of Cardiology

After reperfusion of myocardial infarction (MI), variable recovery of function may occur within "stunned" myocardium (1). Several techniques can assess microvascular integrity or contractile reserve as a means of predicting functional recovery after reperfused MI. Measures of microvascular integrity include contrast echocardiography (CE) (2,3) or coronary flow measured by a Doppler coronary guidewire (4) although this requires catheterization of the infarct-related artery. Noninvasive measures include rest-redistribution thallium (5), technetium-99m sestamibi measures of perfusion and function (6) and gadolinium-diethylenetriaminepenta-acetic acid (Gd-DTPA) enhanced magnetic resonance imaging (MRI) (7–9). Our group has used the latter technique to identify contrast uptake patterns and to correlate them with functional recovery (9). Imaging techniques that examine contractile reserve, or wall thickening in response to inotropic stimulation, to predict functional recovery include dobutamine echocardiography (DE) (10,11) and dobutamine cine MRI (12). Our group has used dobutamine magnetic resonance (MR) tagging (13,14) to demonstrate transmural differences in the predictive value of contractile reserve after reperfused MI (15).

The ability to assess both properties of infarcted myocardium has theoretic advantages over either alone (16) and can be performed with echocardiography or MRI (17). Investigators using echocardiography after MI (18) suggest that CE and DE are complementary. However, image registration between techniques may be problematic, and specificity of CE is limited. The goal of this study was to examine the ability to predict functional recovery using: a) MRI contrast uptake patterns and b) contractile response to dobutamine by tagged MRI within the same myocardial tissue early after reperfused first MI in a single noninvasive examination.

METHODS

Patient population. The Institutional Review Board of Allegheny General Hospital approved the study, and all human subjects gave informed consent. Twenty-seven patients with reperfused first MI were initially enrolled. The diagnosis of MI was made in the conventional manner from clinical history, electrocardiogram and plasma creatine ki-

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Manuscript received December 31, 1999; revised manuscript received May 3, 2000, accepted July 10, 2000.

Abbreviations and Acronyms

CE	=	contrast echocardiography
COMB	=	myocardial regions demonstrating first pass hypoenhancement and delayed hyperenhancement
DE	=	dobutamine echocardiography
Gd-DTPA	=	gadolinium-diethylenetriaminepenta-acetic acid
HYPER	=	myocardial regions demonstrating normal first pass enhancement and delayed hyperenhancement
MI	=	myocardial infarction
MR	=	magnetic resonance
MRI	=	magnetic resonance imaging
%S	=	percent intramyocardial circumferential shortening

nase levels, drawn every 8 h, elevated to more than twice the normal level with MB fraction >5%. All patients underwent coronary angiography and either left ventriculography or two-dimensional echocardiography before enrollment. Patients with recent MI, documented open infarct-related artery after thrombolytic therapy or primary angioplasty with or without stenting and dysfunction in the infarct zone documented by left ventriculography or echocardiography were included in the study. All patients had documented Thrombolysis in MI trial grade 3 flow in the infarct-related artery after therapy. Exclusion criteria included patients with postinfarction angina, active congestive heart failure, atrial fibrillation, aortic stenosis, a history of sustained ventricular arrhythmia, inability to lay flat or standard contraindications to MRI such as pacemakers or cerebral aneurysm clips.

Dobutamine tagging. After an 8-h fast, electrocardiogram-gated MRI was performed on a Siemens (Erlangen, Germany) 1.5T Vision scanner with blood pressure monitoring. All patients were placed supine with the chest within a phased array surface chestcoil. Breath-hold tagged imaging in the left ventricular short-axis from apex to base was then performed (interframe delay 35 ms, repetition time 8 ms, echo time 1 ms, 7 line k-space segmentation, 8 mm thick tag stripes, 7 mm thick slices, 126 × 256 matrix interpolated to 256 × 256 for display, field of view 280 to 300 mm). The tagging method used was a grid-tagging method (14). Signal from protons within the myocardium is nulled in two sets of perpendicular lines at end-diastole, enabling the tracking of intramyocardial points because the signal remains nulled in these tagged lines through the cardiac cycle as the myocardium deforms (Fig. 1). Breath-hold duration was 18 heartbeats (15 s at the average heart rate of the study patients). Dobutamine was infused at 5 and 10 μg/kg/min for a total of 5 min per stage, and apical, midventricular and basal short-axis tagged imaging was performed within the last 2.5 min of each 5-min period as previously described (15) (Fig. 1, left, center).

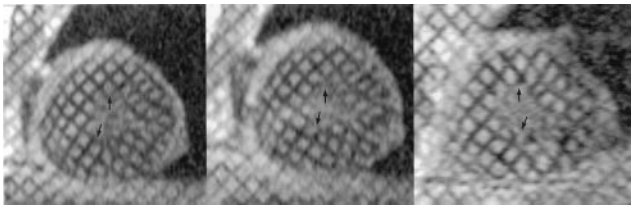

Figure 1. Left panel: End-systolic apical magnetic resonance tagged short-axis image in a patient on day 3 after anterior myocardial infarction treated with percutaneous transluminal coronary angioplasty and stenting of the left anterior descending coronary artery. The septum lies between the arrows, from 7 o'clock to 12 o'clock on the image. Note the reduced deformation of the tag stripes in this region compared with that in the anterolateral wall (12 o'clock to 4 o'clock) and inferoposterior wall (4 o'clock to 7 o'clock). Percent intramyocardial circumferential shortening (%S) was 4% in the septal subendocardium and 3% in the septal subepicardium. **Center panel:** End-systolic apical magnetic resonance tagged short-axis image in the same patient at the end of the 10 μg/kg/min dobutamine stage. Qualitatively, shortening increased significantly in the anterolateral and inferoposterior regions, whereas no improvement is seen in the septum (between the **arrows**). Quantitatively, %S was 5% in the septal subendocardium and 1% in the septal subepicardium, neither of which constitutes improvement. **Right panel:** End-systolic apical magnetic resonance tagged short-axis image in the same patient at nine weeks after myocardial infarction. Function did not improve in the septum (between the **arrows**), either qualitatively or quantitatively, whereas deformation of the tag stripes remains normal in the anterolateral and inferoposterior regions. Percent shortening is 7% in the septal subendocardium and 2% in the septal subepicardium—still significantly depressed. %S = percent intramyocardial circumferential shortening.

Contrast enhanced imaging. Contrast enhanced imaging was performed using an intravenous bolus of 0.1 mmol/kg nonionic Gd-DTPA (gadoteridol, Bracco Diagnostics, Princeton, New Jersey) at 5 cc/s using a power injector (Spectris, Medrad, Indianola, Pennsylvania). T1-weighted inversion-prepared Turboflash images were then sequentially acquired at three locations beginning immediately after contrast injection and lasting for 50 heartbeats. The imaging period for the three slices was 855 ms, the repetition time 3.7 ms, echo time 1.2 ms, inversion time 150 ms, matrix 80 × 128, slice thickness 10 mm and field of view 300 mm. The three slices imaged were two short-axis slices (apex and midventricle for anterior infarctions and midventricle and base for inferior infarctions at the same location as the dobutamine tagged short-axis images) and one long-axis image. At least 1 set of 10 delayed images in each of the same three planes was acquired 5 to 7 min after the first pass imaging described above (Fig. 2). Only the short-axis images were used for analysis for direct comparison with the tagged imaging.

Follow-up imaging. The patients returned 9 ± 1 weeks after the MI for a repeat MRI that consisted of short-axis MR tagging, as described above, without dobutamine. All slices from apex to base were imaged (Fig. 1, right).

Image analysis. Because the first-pass and delayed contrast imaging was performed without a breath-hold, we performed automated in-plane registration and warping of the short-axis images to correct for physiologic motion using an algorithm developed in our laboratory (19). The first pass images and a set of delayed enhancement images (the last if more than one was performed) were reviewed in cine format by two experienced, blinded reviewers for qualitative deter-

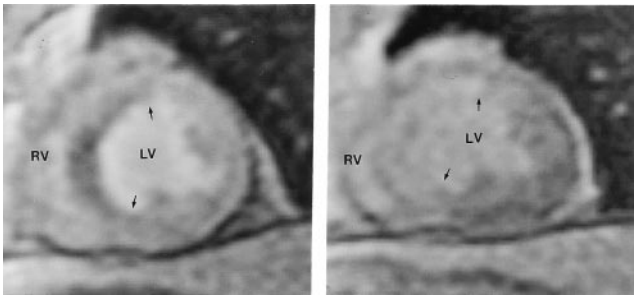

Figure 2. Left panel: Short-axis inversion recovery Turboflash image during first pass of gadolinium-diethylenetriaminepenta-acetic acid through the myocardium in the same patient and the same apical short-axis plane as in Figure 1 with the LV and RV as marked. Note the region of hypoenhancement or reduced signal intensity between the **arrows** in the septum. **Right panel:** Short-axis inversion recovery Turboflash image 5 min after bolus infusion of gadoteridol in same plane as **panel A** with the LV and RV apex as marked. Note the hyperenhancement or increased signal intensity of the septum between the **arrows**. The pattern demonstrated in the two panels of this figure is consistent with that of the COMB pattern (first pass hypoenhancement and delayed hyperenhancement). LV = left ventricle; RV = right ventricle.

mination of abnormal contrast uptake patterns. Interobserver agreement with these techniques and observers was excellent (9). Myocardial regions (quadrants in the short-axis, for example, anteroseptum, anterolateral, etc.) with hyperenhancement through more than two-thirds of the transmural extent of the wall on delayed postcontrast imaging were identified. Within these regions, those demonstrating first pass hypoenhancement, (seen generally in the subendocardial half of the myocardium) were termed COMB. Those demonstrating normal first pass enhancement were termed HYPER.

A blinded investigator (T.M.T.) performed quantitative analysis of the percent of intramyocardial circumferential shortening (%S) using previously published techniques (9,15) and the VIDA software package (University of Iowa, Iowa City, Iowa) on a Sun workstation. Measurements were made on short-axis images within regions defined by abnormal contrast uptake patterns seen on contrast enhanced imaging with two segments (subendocardial or subepicardial) measured per region. A segment within a region was defined as a line defined by a set of tag stripes perpendicular to the endocardial surface in the subendocardium or subepicardium. Percent shortening was measured within segments defined by these tag stripes as end-diastolic minus the end-systolic interstripe distance divided by the end-diastolic interstripe distance times 100 and expressed as a percentage. Therefore, only end-diastolic and end-systolic images were used to measure %S. Measurements were made at baseline and at each dobutamine dose (5 and 10 μg/kg/min). The peak response to dobutamine was recorded. Percent shortening was measured at nine weeks after MI from short-axis slices matched to the day 3 post-MI study by apex to base location, right ventricular insertion sites and papillary muscles as previously performed (9,15).

Statistical analysis. Within each type of contrast defect, %S was compared between baseline, peak dobutamine response and follow-up using repeated measures analysis of variance and Fisher subtesting. Previous work has established a response to dobutamine of $\geq$5% as normal (15,20). The improvement in %S from baseline to follow-up by transmural region was compared by response to dobutamine (normal or abnormal) and by contrast uptake pattern and compared by unpaired *t* test. Linear regression analysis was performed between %S at peak dobutamine and at follow-up. The improvement in function (defined as an increase in %S of $\geq$5% from baseline to follow-up) within regions of the two types of contrast uptake patterns was evaluated. Results are displayed as mean $\pm$ standard error. A p value $<$0.05 was considered to be statistically significant.

RESULTS

Four of the patients did not return for the follow-up MRI. Therefore, 23 patients, 21 of whom were men, with a mean age 51 $\pm$ 2 years completed the study (Table 1). The initial MRI was performed on day 3 $\pm$ 1 after first reperfused acute MI (13 anterior, 10 inferior). Three patients were treated with thrombolytic therapy alone, 3 with thrombolytics followed by rescue angioplasty and stenting, 6 with primary angioplasty and 11 with primary angioplasty and stenting. Peak CPK was 2,824 $\pm$ 466. Twenty-one had Q wave and two had non-Q wave infarctions. All patients had a patent infarct related artery and TIMI grade 3 flow as documented by coronary angiography before enrollment. All had documentation of regional left ventricular dysfunction within the infarct zone by either left ventriculography or two-dimensional echocardiography before enrollment. At the time of the first MR study, all patients were taking aspirin; 19 were taking beta-adrenergic blocking agents; 14 were taking angiotensin-converting enzyme inhibitors, and 7 were taking nitrates.

No patients developed symptoms during or after the dobutamine or the gadoteridol infusion. Heart rate did not change during dobutamine (72 $\pm$ 3 beats/min at baseline, 69 $\pm$ 2 beats/min at 5 μg/kg/dobutamine, 72 $\pm$ 3 beats/min at 10 μg/kg/min dobutamine, p = NS). Likewise, blood pressure did not change; 122 $\pm$ 4/75 $\pm$ 3 mm Hg at baseline, 122 $\pm$ 4/73 $\pm$ 2 mm Hg at 5 μg/kg/min dobutamine, 126 $\pm$ 5/72 $\pm$ 2 mm Hg at 10 μg/kg/min dobutamine, p = NS.

The follow-up study was performed at 9 $\pm$ 1 weeks after the MI. None of the patients had interval cardiac events between the two studies. The heart rate at the time of the 9 week study was 67 $\pm$ 2, and blood pressure was 127 $\pm$ 4/73 $\pm$ 2 (p = NS from baseline study for both). At nine weeks after MI, 21 patients were taking aspirin; 18 were taking beta-blockers; 15 were taking angiotensin-converting enzyme inhibitors, and two were taking nitrates.

Eighty-four transmural regions with an abnormal contrast pattern were identified, and, therefore, function in 168 segments (subendocardial and subepicardial) within these regions was measured. In all 168 segments, %S was 6 $\pm$ 1% at baseline, 8 $\pm$ 1% at 5 μg/kg/min dobutamine, 12 $\pm$ 1% at peak dobutamine (p $<$ 0.0003 vs. baseline and 5 μg/kg/

Table 1. Clinical Information for the 23 Study Patients

Patient #	Age	Gender	Reperfusion	Location	Peak CK	# of Regions
1	43	M	Thrombolysis	Anterior	1,525	4
2	47	M	PTCA	Anterior	1,055	4
3	61	M	PTCA	Inferior	2,460	4
4	54	F	PTCA	Anterior	1,452	4
5	54	M	Thrombolysis	Anterior	3,010	5
6	44	M	Thrombolysis	Inferior	2,172	4
7	39	M	PTCA	Anterior	2,082	3
8	43	M	PTCA	Anterior	4,440	4
9	62	M	PTCA	Inferior	214	3
10	51	M	Thrombolysis	Inferior	3,790	3
11	52	M	PTCA	Inferior	3,655	6
12	48	M	PTCA	Inferior	2,316	2
13	67	M	PTCA	Anterior	4,840	3
14	54	M	PTCA	Inferior	10,150	6
15	49	M	PTCA	Anterior	1,427	4
16	73	M	Thrombolysis	Anterior	3,540	6
17	34	M	PTCA	Anterior	2,556	4
18	41	M	PTCA	Inferior	1,039	3
19	67	M	Thrombolysis	Anterior	6,460	3
20	36	F	PTCA	Inferior	753	2
21	48	M	PTCA	Inferior	4,300	3
22	66	M	PTCA	Anterior	802	4
23	41	M	PTCA	Anterior	897	4

# of regions = number of regions with abnormal contrast uptake patterns. CK = creatine kinase; PTCA = percutaneous transluminal coronary angiography

min dobutamine) and 13 ± 1% at follow-up ($p < 0.0001$ vs. baseline and 5 μg/kg/min dobutamine). When improvement was defined as a 5% increase in %S from baseline to follow-up, 88 of 168 segments improved (52%).

The data as a whole is displayed in Table 2. Within COMB regions, function improved significantly from 4 ± 1% at baseline to 10 ± 1% with peak dobutamine and 10 ± 1% at follow-up ($p = 0.0002$ vs. baseline for both). The HYPER regions demonstrated significant improvement from baseline (10 ± 1%) to peak dobutamine (16 ± 1%, $p < 0.0004$) and follow-up (17 ± 1%, $p < 0.0004$). Percent shortening in HYPER regions was greater than COMB at baseline, peak dobutamine and follow-up ($p < 0.0003$ for each). The absolute increase in %S was similar in both groups from baseline to follow-up (6 ± 1% in COMB and 7 ± 1% in HYPER).

Within subendocardial segments at baseline, %S was higher in HYPER (12 ± 1%) than it was in COMB (5 ± 1%, $p < 0.003$) regions. A similar relationship was noted at peak dobutamine (17 ± 1% vs. 12 ± 1%, $p < 0.01$) and at follow-up (20 ± 1% vs. 11 ± 1%, $p < 0.0001$). Similarly, within the subepicardium, %S in HYPER was greater than it was in COMB regions at baseline (7 ± 2% vs. 3 ± 1%, $p < 0.03$), peak dobutamine (14 ± 2% vs. 7 ± 2%, $p < 0.006$) and at follow-up (13 ± 1% vs. 8 ± 2%, $p < 0.03$).

Of the 76 segments in HYPER regions, 46, or 60%, improved %S at follow-up by 5%, whereas 43 of 92 (47%) segments in COMB regions showed functional recovery at follow-up. The improvement in %S from baseline to follow-up in segments that responded normally to dobutamine ($n = 98$) was 9 ± 1% compared with 3 ± 1% in nonresponders ($n = 70$, $p = 0.0002$). There was a good

Table 2. Percent Circumferential Shortening in the 3 Types of Contrast Defects at Baseline on Day 3, at Peak Dobutamine and at Follow-up on Week 9

		Day 3	Peak Dobutamine	Week 9	ANOVA p Value
COMB	Endo	5 ± 1%	12 ± 1%*	11 ± 1%*	<0.003
(n = 92)	Epi	3 ± 1%	7 ± 2%†	8 ± 2%†	<0.03
	Average	**4 ± 1%**	**10 ± 1%***	**10 ± 1%***	**<0.001**
HYPER	Endo	12 ± 2%‡	17 ± 2%*‡	20 ± 2%*‡	=0.0005
(n = 76)	Epi	5 ± 1%‡	12 ± 1%*‡	12 ± 1%*‡	<0.008
	Average	**10 ± 1%‡**	**16 ± 1%*‡**	**17 ± 1%*‡**	**<0.001**
Total	Endo	8 ± 1%	14 ± 1%*	14 ± 1%*	<0.0001
(n = 168)	Epi	5 ± 1%	10 ± 1%*	10 ± 1%*	<0.001
	Average	**6 ± 1%**	**12 ± 1%***	**13 ± 1%***	**<0.0001**

*$p < 0.001$ versus baseline; †$p < 0.05$ versus baseline; ‡$p < 0.03$ versus COMB.

ANOVA = analysis of variance; COMB = myocardial regions demonstrating first pass hypoenhancement and delayed hyperenhancement; Endo = subendocardium; Epi = subepicardium; HYPER = myocardial regions demonstrating normal first pass enhancement.

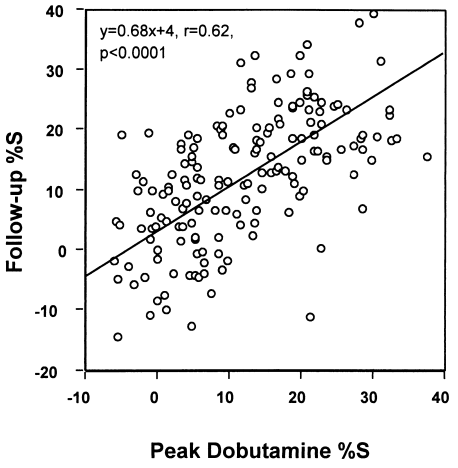

Figure 3. Plot of linear regression analysis for %S at nine weeks after myocardial infarction (follow-up) on the y-axis and at peak dobutamine at day 3 after myocardial infarction for all 168 segments. A significant correlation is demonstrated ($y = 0.68x + 4$, $r = 0.62$, $p < 0.0001$). %S = percent intramyocardial circumferential shortening.

correlation between follow-up %S and %S with peak dobutamine ($y = 0.68x + 4$, $r = 0.62$, $p < 0.0001$; Fig. 3).

DISCUSSION

This study demonstrates that both MRI contrast uptake patterns and contractile response to dobutamine by tagged MRI can be assessed early after reperfused first MI during a single MRI examination. Both techniques impart information about the potential of contractile recovery. Improvement with dobutamine and at follow-up occurred within both COMB and HYPER regions. The COMB regions demonstrated lesser function at baseline, peak dobutamine and follow-up, indicating that the presence of first pass hypoenhancement is associated with greater myocardial damage. Absolute functional improvement in HYPER and COMB were similar. Segments that respond normally to dobutamine (≥5% increase in %S) showed greater late functional recovery than segments that did not. Function at peak dobutamine correlated well with function at rest at nine weeks after MI.

This data extends our group's previous findings regarding the implications of the contrast uptake patterns (9) and a normal dobutamine response (15) for functional recovery. The HYPER region represents predominantly viable myocardium defined as both normal dobutamine response and functional recovery in this setting. The COMB region represents a mixture of viable and nonviable tissue with some late functional improvement. However, the presence of first pass hypoenhancement reflects a greater extent of myocardial injury than seen in HYPER regions. In addition, dobutamine-recruitable function correlates with delayed function at rest.

Comparison with other studies using MRI. Previous work in animal models suggest that contrast uptake patterns reflect Gd-DTPA wash-in and washout kinetics (21). Persistent hypoenhancement represents regions of no reflow (22) and correlate with regions of hypoperfusion on contrast echocardiography (23). In a canine model delayed hyperenhancement reflects infarct size although it overestimates it by 12% (22). In man persistent hypoenhancement has been associated with larger infarction with less adequate reperfusion (8), worse short-term clinical markers (7), lack of functional recovery (9) and worse long-term outcome (24). The findings of delayed hyperenhancement are somewhat more controversial as one study found an association of this pattern with fixed defects on Tl-201 imaging (8) although first pass imaging was not performed, and these may have represented COMB regions. In the setting of chronic ischemic heart disease, delayed hyperenhancement has been shown to be a marker of nonviability, but principally in akinetic or dyskinetic regions at rest (25).

One prior study compared contrast enhanced and dobutamine cine MRI in 28 postinfarction patients (26). In this study no follow-up MRI was obtained to assess late functional recovery, and first-pass uptake of Gd-DTPA was not examined. Delayed imaging was performed 15 min after contrast infusion. In regions with transmural delayed hyperenhancement, no dobutamine response was demonstrated in 10 of 17 (59%) cases. Subendocardial hyperenhancement was associated with a normal dobutamine response in 18 of 22 (82%) regions. Therefore, more transmural hyperenhancement was associated with less dobutamine response. In contrast, in this study, regions with delayed hyperenhancement responded to dobutamine and demonstrated late functional recovery. One potential explanation for the difference is that some of the transmural hyperenhanced regions in the study of Dendale et al. (26) could have been hypoenhanced on first pass imaging had it been performed and, therefore, would have represented COMB regions rather than HYPER regions.

Comparison with echocardiography. Contrast and dobutamine echocardiography have been used together in several studies. In a canine model of reperfused MI, Meza and colleagues (27) demonstrated complementary utility of the two techniques for identifying the absence of necrosis. Sensitivity (88%) for viability was greater than the specificity (61%) of the combined techniques. False positives in the study of Meza et al. were mostly due to the preserved dobutamine response in hypokinetic regions due to subendocardial infarction, as demonstrated in previous work with both echocardiography (28) and dobutamine MR tagging (15).

In 24 patients after MI, Iliceto et al. (18) compared CE with low-dose DE. Contrast echocardiography was 100% sensitive but only 46% specific for the assessment of viability, whereas low-dose DE had a sensitivity of 71% and a specificity of 88%. Dobutamine response and contractile recovery were seen only in the group with preserved contrast enhancement or microvascular integrity, similar to the findings in all regions in this study in which contrast eventually reaches the myocardium (delayed hyperenhancement). Bolognese et al. (29) demonstrated comparable findings in 30 patients after reperfused MI in that microvascular integrity by CE was highly sensitive but nonspecific for functional recovery. Dobutamine echocardiography was

more accurate for prediction of viability (90% vs. 47% for CE). Microvascular integrity is, therefore, necessary but not sufficient for functional recovery. The same is true for the COMB regions in this study in which contrast eventually reaches the microvasculature, but functional recovery is incomplete.

Study limitations. Findings of this study cannot be extrapolated to patients with a residual stenosis in the infarct-related artery, to patients with chronic MI or to patients with less than TIMI grade 3 flow after reperfusion. Only data from two short-axis slices per patient were analyzed. Future improvements in MR perfusion sequences will allow more extensive coverage of the left ventricle. The perfusion imaging was performed without a breath-hold, whereas the tagged imaging was breath-hold, thereby creating a potential matching problem. However, the perfusion images were registered and warped using an algorithm developed in our laboratory, allowing careful matching of landmarks including apex to base location, right ventricular insertion sites and papillary muscles. Matching of the week 9 short-axis tagged images to the early post-MI study is a potential limitation. However, as with previous studies (9,15), careful matching of image locations was used to ensure registration over time.

Acknowledgments

The authors gratefully acknowledge the analytic skills of Jane H. Ripple, RDCS, the support of the MR technologists at Allegheny General Hospital, especially June Yamrozik and Lois Miller, and the patient recruitment efforts of Rebecca Jordan.

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