

VENTRICULAR FUNCTION

Is Post-Systolic Shortening a Reliable Indicator of Myocardial Viability? An MR Tagging and Late-Enhancement Study

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ABSTRACT

Purpose: In ischemic myocardium systolic strain is reduced and followed by a deformation after systole, the so-called post-systolic shortening. The presence of post systolic shortening is therefore considered a marker of viability even though its mechanism remains unclear. The hypothesis was tested whether post-systolic shortening might be a passive recoil phenomenon and therefore not uniquely associated with viability. Methods: Five patients with a history of myocardial infarctions and fully transmural scars in late enhancement imaging and five age-matched healthy volunteers underwent a tagging study to analyze systolic and post-systolic deformation in transmurally infarcted and contra-lateral non-infarcted myocardium. From CSPAMM myocardial tagging data, mid-wall circumferential fiber shortening, radial displacement, and rotation parameters were semi-automatically extracted by harmonic phase (HARP). Results: In transmurally infarcted myocardium, a post systolic shortening of 6.2 \pm 1.8% was present occurring in early diastole (time to maximum circumferential fiber shortening increased versus both, contralateral myocardium and corresponding sectors in healthy volunteers, p < 0.01). Maximum radial displacement was decreased in scar tissue (p < 0.001 versus contra-lateral), but time to maximum radial displacement did not differ. Rotation did not discriminate between infarcted and non-infarcted myocardium. Conclusions: The pure finding of post-systolic shortening is not sufficient for the diagnosis of residual myocardial viability. Post-systolic shortening may be explained in part by passive recoil, which releases energy stored in the scar tissue during systolic intra-ventricular unloading. Circumferential fiber shortening appears best suited for characterization of regional deformation, whereas radial displacement and rotation are more dependent on tethering effects, and thus, are more likely to reflect global chamber mechanics.

INTRODUCTION

During regional myocardial ischemia, a reduced deformation at end-systole (1, 2), and a continued shortening after systole, the

Keywords: Myocardial Tagging, CSPAMM, Cardiac MRI, HARP, Infarct, Post Systolic Shortening. Correspondence to: Juerg Schwitter, MD Division of Cardiology, DIM University Hospital Zurich Raemistrasse 100 8091 Zurich Switzerland phone +41 44 255 3871; fax +41 44 255 4401 email: juerg.schwitter@usz.ch so-called post-systolic shortening (PSS) can be observed (3–6). Despite these experimental findings, the mechanism of PSS is still a matter of debate. Altered local activation or electromechanical coupling, delayed myocardial relaxation, or passive elastic recoil may cause this phenomenon (4, 7). PSS has also been proposed as a marker of viability, and as such could predict recovery of left ventricular function (6). Since viability is an important predictor of outcome (8), we performed the following study to clarify whether PSS is unambiguously related to viability or is caused by passive elastic recoil. Late enhancement (LE) magnetic resonance (MR) imaging is a well accepted method to quantify transmural extent of scar in humans after myocardial infarction (9-11). To address the question of passive recoil as a possible mechanism causing PSS in patients with transmural scar in LE imaging, MR tagging (12, 13) was employed and analyzed using the harmonic phase (HARP) evaluation technique



(a) End-diastolic frame, (b) end-systolic frame. An akinetic inferior sector is visually identified.

(14, 15) to measure systolic and post-systolic shortening in viable remote myocardium and transmural scar tissue. In addition, we were interested in which parameter extracted from tagging data is most powerful to describe the interactions between various myocardial segments, eg, between transmural scar tissue and remote viable myocardium.

METHODS

Study population

In a series of 48 consecutive patients with known coronary artery disease (CAD) who underwent routine LE MR imaging for determination of presence and extent of infarct scar, 5 patients (all male, age: 52 ± 9 years) fulfilled the inclusion criteria for this study of ≥ 1 sector (1 sector corresponds to 1/8 of the circumference Fig. 2) with fully transmural scar and $\geq 40\%$ global left ventricular ejection fraction (This value of $\geq 40\%$ was chosen arbitrarily to recruit patients with sufficient contractile myocardium to generate adequate systolic tension to deform scar tissue). In the patients and in five healthy age-matched volunteers (all male, age: 51 ± 12 years) without a history of CAD and without symptoms or signs of CAD, tagging measurements were performed. For logistical reasons, the tagging measurements were performed on another day than the LE measurements. In the tagging measurement session, no gadolinium-



Figure 2. (a) Late enhancement image acquired 450 ms after the R-wave. An infarct is visible in the sectors 2, 3 and 4. (b) CSPAMM image acquired in the same patient at 675 ms after the R-wave with centerline divided into eight sectors.

chelate was administered. The study protocol was approved by the local ethics committee and written informed consent was obtained from all study participants.

Late enhancement measurements

Following acquisition of SSFP images (8 mm slices thickness, no gap) for measurements of left ventricular volumes and global ejection fraction, 0.25 mmol/kg of Gd-DTPA-BMA (Omniscan, GE Healthcare, United Kingdom) was injected intravenously, and 20 minutes later, LE imaging was performed using a segmented IR-GRE sequence with inversion time set to null normal myocardium (FOV: $380 \times 285 \text{ mm}^2$, matrix: 224×160 , TR: 6.4 ms, TE: 1.6 ms, slice thickness 8 mm, no gap, same location as for cine SSFP acquisitions) (11). The images were acquired on a 1.5 T MR scanner (Twin Speed, GE Medical Systems, Milwaukee, WI, USA)

CSPAMM tagging measurements

A complementary spatial modulation of magnetization (CSPAMM) (13) tagging approach was used to allow for slice following (ie, through-plane tracking of myocardium during contraction and relaxation) and to minimize tag fading during the cardiac cycle on a 1.5 T MR scanner (Gyroscan Intera, Philips Medical Systems, Best, The Netherlands). Two CSPAMM images with orthogonal one-dimensional stripe patterns with 8 mm tag distance were acquired in five short-axis slices (slice-thickness: 8 mm, gap: 15 mm). Imaging parameters of the EPI sequence were as follows: FOV: $380 \times 304 \text{ mm}^2$, TE: 7.5 ms, TR: 15 ms, EPI-factor: 13, matrix: 128×39 , final flip angle: 25° . Sixteen to twenty frames were imaged with a temporal resolution of 31–35 ms.

Data analysis

The CSPAMM images were evaluated with peakcombination HARP (15) to correct for phase errors arising, eg, from B₀-inhomogeneities, a modification of HARP (14). The endocardium and epicardium were manually selected on the image from the latest acquired end-diastolic frame (16), where the endocardial border is best visualized (Fig. 2B). Points were automatically placed in steps of 5° on these epi- and endocardial circumferences and the centerline was calculated as the middle of these border contours at end-diastole. The centerline was then divided circumferentially into eight equal sectors (Fig. 2B), relative to the position of the anterior junction of right and left ventricle (reference point) and tracked over all cardiac frames. Circumferential fiber shortening of the centerline (cFS in %) was calculated over the cardiac cycle for each sector by:

$$cFS(s,hp) = \left(1 - \frac{CL(s,hp)}{CL(s,0)}\right) \cdot 100\%$$
[1]

where s indicates the sector, hp indicates the cardiac frame, and CL indicates the length of the centerline.

Radial displacement and rotation were calculated with respect to the center of gravity of the centerline. The radial displacement (RD) of each cardiac frame (hp) is:

$$RD(s,hp) = \left(1 - \frac{\sum_{p(s)} |\vec{r}(p,hp)|}{\sum_{p(s)} |\vec{r}(p,0)|}\right) \cdot 100\%,$$
 [2]

where $\vec{r}(p,hp)$ is the vector from the dynamic center of gravity (cg) to the point (p) on the centerline.

The rotation angle, $\varphi(p,hp)$, was defined as the angle between $\vec{r}(p,hp)$ and $\vec{r}(p,0)$ averaged over all p in the sector s:

$$m(p,hp) = \frac{y(p,hp) - y(cg,hp)}{x(p,hp) - x(cg,hp)}$$
[3]

and

$$\varphi(\mathbf{p},\mathbf{hp}) = \arctan\left(\frac{\mathbf{m}(\mathbf{p},\mathbf{hp}) - \mathbf{m}(\mathbf{p},0)}{1 + \mathbf{m}(\mathbf{p},0) \cdot \mathbf{m}(\mathbf{p},\mathbf{hp})}\right).$$
 [4]

The slope of the contraction or stretching is the maximum or minimum cFS divided by the time of the contraction/stretching in seconds.

In order to correct for different heart-rates the shortening-, displacement- and rotation-curves were normalized. In each data set, the cardiac frame with the minimal circumference of the LV-midline was determined and defined as end-systolic frame (100% ES). Each deformation curve (cFS, RD, rot) was subsequently resampled at 20 time-points, where the end-systolic frame was set to the ninth frame for all data sets. These resampled curves were only used for illustration purpose, the deformation values for the statistical analysis were extracted from the original data sets.

On the late-enhancement images, the transmurally infarcted sectors were identified. cFS was compared between these infarcted and the non-infarcted contra-lateral sectors. If more than one sector was infarcted, only the main affected sector was used for further evaluation (eg, sector 3 in the case shown in Fig. 2A). The same sectors were also evaluated on the data of the volunteers.

Statistics

The extracted deformation parameters of the infarcted sectors and the contra-lateral sectors in patients (pat_{inf} and pat_{cl}) and the corresponding sectors in the volunteers (vol_{c_inf} and vol_{c_cl}) were compared with an analysis of variance (ANOVA) for repeated measurements followed by Bonferroni post-hoc testing (InStat, 3.01, Graph-Pad Software Inc., San Diego, USA). P values <0.05 were considered statistically significant.

RESULTS

The SSFP short axis images in Figure 1 show an akinetic inferior sector with Figure 2A demonstrating the late-enhancement data of the same patient with an infarct in this inferior sector (sector 3). The contraction was evaluated on the corresponding CSPAMM data (Fig. 2B), leading to the cFS curves as displayed in Figure 3. Sector 3 shows a different contraction pattern than



the contra-lateral sector 7. A stretching of the region is observed during systole whereas shortening occurs during diastole causing PSS.

Patient characteristics are given in Table 1. Deformation parameters averaged over all patients are displayed in Figure 4 for infarcted sectors and the corresponding contra-lateral sectors. As in the example in Figure 3, the averaged cFS curve on Figure 4A shows a stretching of the infarct sector during systole followed by PSS. RD does not show a different pattern of contraction between scar and contra-lateral sectors. But overall RD is decreased in the infarcted sectors by approximately a factor of 3 compared to the contra-lateral sectors (Fig. 4B). The rotation curve does not seem to differentiate between infarcted and contra-lateral sectors (Fig. 4C).

Table 2 shows the maximum values of the evaluated deformation parameters. In patients, maximum cFS in the infarct sectors (pat_{inf}) occurred in early diastole (=PSS, 6.2%, p < 0.01 vs. 0) and was decreased compared to the contra-lateral sectors in patients (pat_{cl}, p < 0.01) and the corresponding sectors in the volunteers (vol_{c_inf}, p < 0.001; Fig. 5). Conversely, the time until maximum cFS was increased in pat_{inf} (p < 0.01 vs. both pat_{cl} and vol_{c_inf}). Maximum RD was also decreased for pat_{inf} compared to pat_{cl} and vol_{c_inf} (p < 0.001 for both) but the time to maximum RD did not differ. There was also no significant difference between the maximum rotation of pat_{inf} and pat_{cl} or

Table 1. Clinical characteristics of patients

Patient	Age (years)	Diagnosis	Treatment	LV-EF (%)	LV mass (g)	Scar mass (% LV mass)
1	49	Anterior MI + PCI + 2x in-stent thrombosis	BB, ACEI, AC	40	169	26.3
2	46	Silent antero-septal MI	BB, ACEI, nitrates	43	128	22.7
3	57	Infero-posterior MI, recurrent infero-lateral MI + lysis	BB, ACEI, diu, amio	41	139	20.4
4	65	OHT (dilative CMP)	Immunosuppression	55	116	16.3
		Antero-septal MI + PCI (stenting)	BB, AT ₁ -blocker, diu, AC			
5	41	Infero-lateral MI + lysis + reconstructive RCA surgery aneurysms in RCA, LCX, ectatic LAD	BB, ACEI	63	132	14.6

All patients were on platelet aggregation inhibitors and statins, when appropriate.

MI = chronic myocardial infarction; VF = ventricular fibrillation; PCI = percutaneous coronary intervention; CMP = cardiomyopathy; OHT = orthotopic heart transplantation; LAD = left anterior descending coronary artery; LCX = left circumflex coronary artery; RCA = right coronary artery; BB = beta-blocker; ACEI = angiotensin-converting enzyme inhibitor; AC = anticoagulation; diu = diuretics; $AT_1 =$ blocker: Angiotensin-receptor blocker; amio = amiodarone LV-EF = left ventricular ejection fraction.

 vol_{c_inf} . The slopes, characterizing the stretching in the infarct sector and the contraction in the contra-lateral sector, differed significantly (p < 0.001 vs. pat_{cl} and vol_{c_inf}). In the volunteers, none of the deformation parameters differed between vol_{c_inf} and vol_{c_cl} . Also, none of the deformation parameters of pat_{cl} differed vs. vol_{c_cl} .

DISCUSSION

The current study suggests that in transmural scar tissue, ie, in the absence of viable tissue, PSS does occur as assessed by CSPAMM tagging and HARP analysis. Consequently, PSS is not unambiguously related to viability.

Assessment of systolic deformation in transmural scar tissue and contra-lateral viable myocardium

In patients after myocardial infarction, cFS at the end of systole was absent in transmural scar tissue and was followed by a PSS of 6.2%, which occurred 198 ms after aortic valve closure (Fig. 5). This feature was strikingly different from normal vi-

Table 2. Deformation parameters are compared for the infarcted and contra-lateral sectors in patients (pat_{inf} and pat_{cl}) and the corresponding sectors in healthy volunteers ($vol_{c.inf}$ and $vol_{c.cl}$)

	pat _{inf}	pat _{cl}	vol _{c_inf}	vol _{c_cl}
max.cFS [%]	$\textbf{6.2} \pm \textbf{1.8}$	$20.0\pm5.6^{\ast}$	$20.2\pm3.1^{\ast}$	24.5 ± 3.2
max.RD [%]	$\textbf{7.0} \pm \textbf{2.4}$	$17.0\pm5.9^\dagger$	$22.1\pm2.3^{\ast}$	23.2 ± 2.2
max. rotation [deg]	$\textbf{6.2} \pm \textbf{1.6}$	5.9 ± 3.2	$\textbf{6.5} \pm \textbf{3.1}$	5.6 ± 2.6
slope 1st extr. [%/s]	-65 ± 53	$62\pm19^*$	$64\pm9^*$	81 ± 21
max cFS time [ms]	458 ± 65	$325\pm58^\dagger$	$334\pm55^\dagger$	326 ± 40

Parameters in pat_{cl} were not different from corresponding sectors in volunteers vol_{c_cl} and none of the parameters differed for vol_{c_cl} vs. vol_{c_inf} . (Statistics were not performed for pat_{cl} vs. vol_{c_inf} and pat_{inf} vs. vol_{c_cl} .)

Deformation parameters are indicated as mean \pm 1SD, $^{\dagger}p <$ 0.01; $^{*}p <$ 0.001.

able myocardium of controls, which showed a peak cFS at end of systole of 19.5%, while PSS did not occur. cFS further differentiated transmural scar tissue from normal myocardium by a stretching, ie, a negative cFS, during early systole. Thus, cFS revealed a fundamentally different deformation pattern of transmural scar tissue vs. remote and normal viable myocardium with initial stretching of the scar tissue without a measurable cFS at end-systole but followed by a mild PSS. This pattern was not observed for RD of scar tissue, which showed a mild centrifugal displacement of 7% after end-systole, but no stretching or outward displacement in early systole. Finally, torsion or twist of the LV myocardium was shown to be a sensitive marker of myocardial damage (17). However, in the current study, the rotation parameter proved to be insensitive for viability on a regional basis since it did not differentiate sectors with transmural scar from viable normal sectors. Also, no differences in rotation were found between patients and volunteers. This may be explained by the fact, that rotation was assessed at the mid-ventricular level for most patients, where rotation is known to be minimal even in healthy hearts (18). Together, these data suggest, that cFS is a useful parameter to characterize and quantify local myocardial deformation, while RD, and particularly rotation, are less sensitive for local alterations in deformation. One explanation might be that RD and rotation are more susceptible for tethering effects and thus, are more likely to reflect global chamber mechanics. Similarly, tissue Doppler imaging in echocardiography is prone to tethering effects, whereas strain and strain-rate imaging are more closely reflecting local deformation comparable to cFS in MR tagging.

From tagging data, a large variety of different functional parameters can be derived. cFS, RD, and torsion were validated with respect to rest and stress conditions and reproducibility in a previous study (16). Other investigators looked at minimum and maximum principal strain and the angle between them (19). These parameters, in particular the angle between strains indicated that increased shear between endo- and epicardial layers can affect contractile function of remote myocardium. For future studies using CSPAMM and peak-combination HARP



sectors.

it appears worthwhile to also validate and assess intramyocardial shear parameters to better understand intramyocardial mechanics.

Mechanisms of PSS

The fact that post-systolic deformation occurred in transmural scar tissue as confirmed by LE imaging strongly supports the notion that this post-systolic deformation is caused by a passive recoil phenomenon. In a study using echocardiographic strainrate imaging in pigs (20) with acute infarctions, end-systolic shortening of 3% increased slightly to a PSS of 9%, whereas in non-transmural infarctions, end-systolic shortening of 32% increased further to 45% PSS. Hence, in this animal experiment, PSS was 6% in transmural scar and 13% in non-transmural scar. The current study in humans is in agreement with these animal data with a PSS of 6% in complete transmural scar. Slightly higher PSS values of 7.2–11.7% were reported for acute ischemia in pigs using echocardiographic myocardial strain imaging (21), while MR tagging of transmural scar in dogs yielded a peak cFS of 2.5% (22). In patients with CAD, PSS during ischemia was 6.7% as assessed by echocardiographic myocardial



strain imaging (during dobutamine stress) and was the best parameter for detection of ischemic dysfunction (23). These findings are indicative that PSS in ischemic tissue is similar or only slightly higher than in scar tissue and passive recoil is likely to play a role during "ischemic" PSS as well. Smalling et al. described a reciprocal deformation pattern between ischemic and remote myocardial regions in an acute ischemia model in dogs, which was explained by intra-ventricular unloading (3). Based on the results of the present study, intra-ventricular unloading causing a passive recoil of transmurally infarcted regions postsystole is a likely mechanism involved in PSS.

In the acute animal study by Smalling et al. (3), the remote regions showed hyper-contractions. These may be related to the acute onset of ischemia in that model. In the present study in patients with chronic infarctions, no hyper-contractions of the remote myocardium were observed. A long-standing loss of local function in the infarcted regions is likely to provoke remodeling, ie, compensating hypertrophy in the remote regions and several studies showed a reduced cFS in hypertrophied myocardium (12, 24). Compensatory hyper-contraction of remote regions was also missing in the chronic state in pigs (20). Similarly, in patients 8 weeks after acute anterior myocardial infarctions, remote myocardium showed normal, but not hypercontractile function as assessed by MR tagging (25), while in the acute phase, both infarcted and remote myocardium showed impaired contractile function (26). Furthermore, all patients in the current study were on beta-blockers, which may further contribute to a lack of regional hypercontractile function in remote myocardium (although beta-blockers can improve global LV ejection fraction most likely through LV remodeling).

CONCLUSIONS

In patients with chronic myocardial infarctions, a small amount of PSS does occur in transmural scar tissue indicating that the pure finding of PSS is not sufficient for the diagnosis of residual viability. PSS may be explained in part by passive recoil of transmural scar tissue which releases energy stored in the scar tissue during systolic intra-ventricular unloading.

cFS appears most suitable to assess reciprocal functional interactions of myocardial sectors, whereas RD and torsion are more dependent on tethering effects and, thus, are more likely reflecting global chamber mechanics. Therefore, cFS may be a helpful parameter to assess regional dyssynchrony in patients with impaired contractile function.

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