Myocardial Strain and Torsion Quantified by Cardiovascular Magnetic Resonance Tissue Tagging
Studies in Normal and Impaired Left Ventricular Function

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Accurate quantification and timing of regional myocardial function allows early identification of dysfunction, and therefore becomes increasingly important for clinical risk assessment, patient management, and evaluation of therapeutic efficacy. For this purpose, the application of tissue Doppler echocardiography has rapidly increased. However, echocardiography has some major inherent limitations. Cardiovascular magnetic resonance imaging with tissue tagging provides highly reproducible data on myocardial function, not only in longitudinal and radial directions, but also in the circumferential direction. Because of the development of faster imaging protocols, improved temporal resolution, less time-consuming postprocessing procedures, and the potential of quantifying myocardial deformation in 3 dimensions at any point in the heart, this technique may serve as an alternative for tissue Doppler echocardiography and is now ready for more widespread clinical use. This review discusses the clinical use of cardiovascular magnetic resonance tissue tagging for quantitative assessment of regional myocardial function, thereby underlining the specific features and emerging role of this technique. (J Am Coll Cardiol 2006;48:2002–11) © 2006 by the American College of Cardiology Foundation

Development of new methods of accurate quantification of regional myocardial function have been urged by the clinical need to predict contractile recovery in patients with ischemic heart disease (1), and recently developed novel therapeutic options for improving local function, such as stem cell therapy (2) in patients with heart failure and percutaneous transluminal septal myocardial ablation in patients with hypertrophic obstructive cardiomyopathy (3). Timing of regional myocardial function is essential for optimizing selection of candidates for cardiac resynchronization therapy (CRT) (4).

Strain analysis allows a direct assessment of the degree of regional myocardial deformation and its timing during the cardiac cycle. Strain is expressed as the percentage of shortening or lengthening of a small element of myocardium in relation to its original length. Both echocardiography with tissue Doppler imaging (5–7) and cardiovascular magnetic resonance (CMR) tissue tagging (8,9) are imaging techniques capable of measuring myocardial strain.

The use of tissue Doppler echocardiography for strain quantification has rapidly increased, especially in the setting of resynchronization therapy (10,11). Echo-Doppler derived parameters are predominantly based on longitudinal and radial motion data. However, myocardial contraction is principally circumferential (12,13), and thus echo-based Doppler methods do not allow the most accurate evaluation of myocardial function. Cardiovascular magnetic resonance imaging provides highly reproducible data of myocardial deformation, not only in longitudinal and radial directions, but also in the circumferential direction. Because of the development of faster imaging protocols and improved temporal resolution (14,15), less time-consuming postprocessing procedures, and the potential of quantifying myocardial deformation in 3 dimensions at any point in the heart (16,17), CMR tissue tagging has become a powerful tool for basic research and seems ready for more widespread clinical use.

Several studies have been published in the past using CMR tissue tagging. In this review we focus on the clinical applications of CMR tissue tagging for evaluation of normal and diseased myocardium, thereby underlining the specific features and emerging role of this technique for assessment of regional myocardial function.

CMR TISSUE TAGGING

In 1988, Zerhouni et al. (8) introduced a new CMR technique to magnetically label or tag different regions
within the heart wall. The basic idea was to create noninvasive markers within the heart wall by applying saturation planes perpendicular to the imaging plane at the electrocardiographic trigger signal before image acquisition. During the subsequent image acquisition, reduced signal is obtained from the saturated tissue. Therefore, the cut line of the image plane and the saturated plane appears as a hypointense or black line on the images (Fig. 1).

A fast way to generate a tagging pattern was introduced by Axel and Dougherty (9) in 1989. This method is known as spatial modulation of magnetization (SPAMM). Because SPAMM is fast, it can be applied twice, in 2 orthogonal directions, yielding a grid pattern. With SPAMM either sharper stripes or a sinusoidal intensity variation can be obtained (18).

Because the magnetization is a property of the tissue, the tag lines move along with the tissue in which they are created. When created at end diastole (Fig. 2A), the lines will deform with the myocardium during contraction (Fig. 2B), and become undeformed again during subsequent relaxation (Figs. 2C and 2D). From this it can be recognized that the deformed tag pattern reflects the underlying motion of the heart wall. By tracking the motion of the tag lines throughout the cardiac cycle, the intramural myocardial deformation can be quantified (Fig. 3).

The tags gradually will fade during the cardiac cycle (Fig. 2) because of tissue T1 relaxation and the imaging radiofrequency pulses. This fading may hamper assessment of regional myocardial function, especially the analysis of the relaxation of the heart during diastole.

To improve the tag contrast relative to the background of the relaxed, nontagged signal, Fischer et al. (19) introduced the use of complementary spatial modulation of magnetization (CSPAMM). With CSPAMM, the tagging signal is separated from the relaxed signal by subtracting 2 measurements, one with a positive and one with a negative tagging pattern. A disadvantage of CSPAMM is the increased image acquisition time. Further improvement can be obtained by choosing imaging sequences such as echo planar imaging (20) or balanced steady-state free precession (15,21). For a complete overview and evaluation of the different pulse sequences that can be used in combination with tagging, we refer to the excellent review by Axel et al. (22).

**Tagged image analysis.** The analysis method used to extract information on myocardial function from the tagged images depends on the clinical purpose for which it is applied.

Visual assessment requires tags that can be easily followed by eye, and therefore the tag lines should be as sharp as possible. For this purpose image quality is essential, whereas temporal resolution and the applicability of semiautomatic postprocessing become less important. This means for the image acquisition that a rectangular intensity profile of the tag lines is preferred, and that grid-tagged images are presented to the observer. A SPAMM with a sharp grid is one of the most widely used techniques for this purpose.

### Abbreviations and Acronyms

- **CMR** = cardiovascular magnetic resonance
- **CRT** = cardiac resynchronization therapy
- **CSPAMM** = complementary spatial modulation of magnetization
- **DCM** = dilated cardiomyopathy
- **HARP** = harmonic phase
- **HCM** = hypertrophic cardiomyopathy
- **LV** = left ventricle/ventricular
- **SPAMM** = spatial modulation of magnetization

**Figure 1.** Basic principle of magnetic resonance tissue tagging. Cardiovascular magnetic resonance tissue tagging. The schematic drawing indicates the short-axis image planes and the magnetically saturated planes perpendicular to the imaging plane (A). During subsequent image acquisition, reduced signal is obtained from the saturated tissue. This results in black lines on the images (B). LV = left ventricle; RV = right ventricle.
For quantitative assessment of myocardial deformation with (semi)automated analysis methods, however, other types of tagging images may be more suitable. Basically, 3 types of analysis methods can be discerned. The first type of method aims at the detection and tracking of the tag lines in the images. Software programs such as Findtags (23) and SPAMMVU (24) are based on methods of this type.

The second type is based on optical flow, a method originating from machine vision applications, which determines the motion of the object in the image by assessing the images. Software programs such as Findtags (23) and SPAMMVU (24) are based on methods of this type.

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Figure 2. Example of short-axis tagged images during cardiac cycle. Typically, tagging is applied just before contraction starts (end diastole, A). Because the taglines are a temporary property of the tissue, taglines move along with the tissue in which they are created. The lines will deform if the myocardium contracts (B), and become undeformed during relaxation (C). Because of T1 relaxation, the contrast between tagged (dark) and nontagged (bright) myocardium will gradually disappear during subsequent image acquisition (D). LV = left ventricle; RV = right ventricle.

Figure 3. Representation of myocardial deformation using magnetic resonance tissue tagging. An example of a short-axis image with deformed tagging grid on end systole is shown (A). Tagging is achieved by the application of spatial modulation of magnetization (SPAMM) (18). The tag-tag distance is 7 mm. Using the SPAMMVU software package, the intersection points of the tagging grid can be marked (B) and tracked throughout the cardiac cycle (C). The displacement of these points can be measured, and subsequently the intramural myocardial deformation can be quantified.
temporal and spatial changes in image intensity. The third type is the harmonic phase (HARP) analysis (25). The HARP method calculates for each pixel the spatial phase in the periodic tagging pattern. This phase can be used to track the points through the cardiac cycle, or to calculate the deformation directly, by calculating the regional spatial frequency of the tagging pattern and comparing it with the undeformed frequency. For a more in-depth review and further references on image analysis, we refer again to Axel et al. (22).

Because of its almost fully automated nature, HARP is currently the most widely used analysis method. It has been shown that the use of CSPAMM yields significantly better strain results when HARP analysis is used (26). Although traditionally the HARP method is optimized for high temporal resolution and low spatial resolution, and displacement-encoded imaging with stimulated echoes for low temporal resolution and high spatial resolution, this is not an essential difference intrinsic to the either method. Strain analysis. Strain analysis describes the change of shape of material (i.e., myocardium) resulting from deformation. Strain is independent of rigid body motion (translation and/or rotation) and does not necessarily need an external reference system.

Principal and normal strains. In published reports, principal strains as well as normal strains are used to present regional myocardial function. The principal or maximum strains provide the maximum deformation in 3 orthogonal directions, irrespective of the geometry of the heart (27,28). For a meaningful clinical interpretation, the orientation of the maximum stretch should also be taken into account.

For interpretation of normal strains, a coordinate system is required. Two different coordinate systems, the radial/fiber/cross fiber system (29,30) and the radial/circumferential/longitudinal system (31,32), are used for this purpose. For the radial/fiber/cross fiber systems, the fiber orientation is required. For clinical purposes, description of strain in the radial/circumferential/longitudinal system is preferable. Using this coordinate system, myocardial deformation can be described in a more intuitive way (Fig. 4): 1) Radial strain, representing deformation of the heart in the radial direction (i.e., toward the center of the ventricle). Positive radial strain during systole reflects the local contribution of the myocardium to wall thickening, and negative radial strain during systole implies local wall thinning (dyskinesia); 2) Circumferential strain, reflecting intramural circumferential shortening. A negative value is related to myocyte contraction, and a positive value indicates systolic bulging; and 3) Longitudinal strain, reflecting the regional amount of myocardial shortening from base to apex (negative value).

Strain rate. The strain rate can be derived from the different strain measures by dividing strain by the time information (T) from each time frame:

\[
\text{Strain rate} = \frac{\text{Strain} \, (t_2) - \text{Strain} \, (t_1)}{t_2 - t_1}
\]

Time derivative of strain is the strain rate (1/s) and is of importance in assessing the relaxation of the ventricle. Quantification of the strain rate allows detailed evaluation of left ventricular (LV) diastolic function (33,34).

Shear strains. In addition to the normal strains, 3 shear angles can be calculated: the radial-circumferential shear angle \( \alpha_{rc} \), the radial-longitudinal shear angle \( \alpha_{rl} \), and the circumferential-longitudinal shear angle \( \alpha_{cl} \). The circumferential-longitudinal shear angle is the most interesting shear angle: it describes the twisting motion of the heart and is closely related to torsion of the LV.

**Torsion.** Cardiovascular magnetic resonance tissue tagging also offers the opportunity to quantify myocardial torsion. The twisting motion of the LV stores potential elastic energy by straining the extracellular matrix, which is released during early diastole, and therefore probably contributes to early diastolic suction (35). In the LV, torsion and untwisting is the result of contraction and relaxation of the spiraling myofibers (36). Several different definitions are used to describe torsion (37,38). However, the most widely used definition is the circumferential-longitudinal shear on the epicardial surface between 2 short-axis slices (Fig. 5).
STUDIES IN THE NORMAL LV

Myocardial strain in the healthy subject. In the normal heart, the end-systolic circumferential and longitudinal strains (shortening) gradually increased from base toward the apex and from epicardium toward the endocardium. For the end-systolic radial strain (representing the local contribution to wall thickening), significant differences among slices were not found. However, this finding on radial strain may be partly a consequence of the lowest spatial density of tag data in the radial direction, and thereby a less accurate measurement of strain in the radial direction (39).

In another study, the circumferential, longitudinal, and radial strains increased from base to apex. Also between segments differences were found: the highest absolute values were found for radial strains in the anterior and septal regions, and for circumferential and longitudinal strains in the posterior and lateral walls. Despite the high radial strains in the anterior and septal regions, the regional ejection fraction in this area was smaller, probably as a result of the lower circumferential and longitudinal strains in this part (40). These observed regional differences in strain are probably related to regional differences in regional LV architecture, function, and local stress.

When tagged images are obtained with high temporal resolution (14 ms), strain analysis allows detailed mapping and timing of myocardial deformation. In healthy subjects, both the onset time of circumferential shortening (T_{onset}) in early systole and the time of peak circumferential shortening (T_{peak}) at end systole were studied. The onset of shortening width (time needed for 20% to 90% of the LV to start shortening) was small (35 ± 9 ms). A distinct spatial pattern for T_{onset} was found, with earliest onset in the lateral wall and latest onset in the septum (p = 0.001). Compared with T_{onset}, the T_{peak} had a larger width (121 ± 22 ms) and an opposite spatial pattern, with peak shortening occurring earlier in the septum than in the lateral wall (p < 0.001).

These maps may serve as a reference in detecting mechanical asynchrony because of intraventricular conduction delays or ischemia (41).

MYOCARDIAL STRAIN IN RELATIONSHIP TO AGE

Three-dimensional MR tissue tagging was applied by Fonseca et al. (42) and Oxenham et al. (43) to determine the effects of aging on regional LV myocardial contraction and relaxation. Strain analysis was performed in 15 younger (age 19 to 26 years) and 16 older (age 60 to 74 years) healthy volunteers.

In the older group, peak apical rotation and torsion were increased during systole and persisted longer. The peak rate of untwisting was reduced in older subjects (74 ± 16°/s vs. 91 ± 15°/s, p < 0.01), and this reduction of the untwisting rate was larger at the base than at the apex (p < 0.05). These observations show that patterns of regional nonuniformity of diastolic myocardial function (including prolonged systolic torsion and reduced untwisting rate) are associated with aging.

STUDIES IN THE IMPAIRED LV

Coronary artery disease. After transmural myocardial infarction, changes in intramural myocardial function will occur. Independent of localization, intramural deformation was found to be reduced in the infarcted myocardium. Therefore, the infarct area can be recognized by a specific spatial pattern of intramural deformation, including reduced and more circumferentially oriented systolic stretch (local systolic dilatation or bulging). Regional differences in intramural deformation between infarcted and noninfarcted (remote) myocardium were larger in the left anterior descending coronary artery related infarcts than circumflex or right coronary artery related infarcts of enzymatically the same size (44). This finding may be one of the mechanical
explanations of why anterior myocardial infarction seems more prone to postinfarct remodeling (45).

In a direct comparison between strain and wall thickening analysis, strain analysis was found to be superior in discriminating infarct from remote myocardium. In this study, the perfusion territory of the culprit vessel was considered the infarct region. For detecting dysfunctional myocardium, wall thickening analysis had a sensitivity of 69% and a specificity of 92%, whereas strain analysis showed a sensitivity of 92% and a specificity of 99%. The global ejection fraction correlated better with averaged myocardial strain ($r = 0.89, p < 0.001$) than with wall thickening ($r = 0.76, p < 0.005$) (46).

Some controversy exists about myocardial function in noninfarcted remote myocardium. In one study performed within 10 days after infarction, dysfunctional regions (infarcted myocardium), regions with normal function (adjacent myocardium), and regions with hypercontractile function (remote myocardium) were observed (47).

Another study, however, showed that patients with single-vessel disease had reduced intramyocardial circumferential shortening throughout the LV, including the remote noninfarcted regions on day 5 after first anterior infarction (48). One explanation for these different findings might be the difference in definition of remote areas.

Eight weeks after first anterior infarction, strains remained reduced not only at the infarct area, but also in the remote myocardium of the noninfarcted basal segment as well (49). This persistent dysfunction in infarcted and noninfarcted areas may be one of the first indicators of progressive remodeling and the occurrence of heart failure late after myocardial infarction.

Ischemia also affects LV torsion and untwisting. In animal experiments, ischemia induced by a short period of coronary artery occlusion resulted in increased counterclockwise rotation. A probable explanation is the loss of counteraction of contraction of subendocardial, clockwise-oriented fibers because of subendocardial ischemia. When the occlusion persisted, the torsion pattern showed a form of pseudonormalization and finally was globally decreased, because contractility of subepicardial fibers also decreased (50). A decrease in torsion was also observed in patients after myocardial infarction (51). This reduction of torsion was related to the extent of the asynergic area and to a decrease of global ejection fraction.

**CAD and response to dobutamine stress.** Dobutamine stress echocardiography is an established technique for studying the presence of viability and ischemia (52). Several studies have shown that dobutamine MR tissue tagging is as safe and effective in detection of wall motion abnormalities, and therefore is a good alternative to dobutamine stress echocardiography.

A comparison between low-dose dobutamine stress echocardiography and MR tissue tagging (infusion of 5 and 10 μg/kg/min dobutamine) was performed by Kramer et al. (53). The investigators showed that both techniques are sensitive and accurate for the prediction of functional improvement after reperfused myocardial infarction using a 5% increase in intramyocardial circumferential shortening at peak response to dobutamine as evidence of normal contractile reserve (54). The sensitivity and specificity of dobutamine MR tissue tagging were 82% and 69%, respectively, with an overall accuracy of 76%. Using the same threshold for response of dysfunctional myocardium to dobutamine, functional recovery 8 weeks after myocardial infarction could be predicted ($p < 0.04$). Dysfunctional myocardium that does not respond normally to peak dobutamine did not show functional improvement (55).

In a study by Kuipers et al. (56), more than 200 patients with chest pain were studied with high-dose dobutamine CMR tissue tagging. This study showed that high-dose dobutamine CMR tissue tagging detected more new wall motion abnormalities (68 of 211 patients) than dobutamine CMR studies without tagging (58 of 211 patients, $p < 0.01$). Coronary angiography confirmed the presence of coronary artery disease, defined as a diameter reduction of >50% in 1 or more major epicardial coronary arteries, in 65 (96%) of the 68 patients identified by CMR tissue tagging, of which 62 patients needed revascularization. The sensitivity of dobutamine MR tissue tagging for detecting significant coronary artery disease was 92%, and the specificity was 98%.

**Dilated cardiomyopathy.** In patients with nonischemic dilated cardiomyopathy (DCM), a consistent pattern of marked regional heterogeneity in myocardial function was found. In the septum, systolic lengthening (in both circumferential and longitudinal direction) instead of shortening was found during systole, whereas in the lateral wall, relatively normal systolic shortening occurred ($p < 0.001$ lateral vs. septal walls). Reduced function in the septal region may be related to increased wall stress, leading to relative hypoperfusion and subsequently myocardial dysfunction (57).

In general, subendocardial hypoperfusion and concomitant reduced contraction in the subendocardium will lead to limited counteraction of clockwise rotation of the subepicardial fibers. This results in an increased net ventricular torsion. Importantly, decreased contractility and associated increased torsion as a result of hypoperfusion may occur far before irreversible tissue damage including increased collagen content and altered architecture becomes present (58–60), and thus may serve as early indicators of (reversible) cardiac abnormalities in patients prone to the development of structural cardiac damage and dysfunction.

Another factor contributing to heterogeneity in regional myocardial function in these patients might be the presence of conduction abnormalities. Dilated cardiomyopathy is often complicated by intraventricular conduction delay (~25%), usually manifested as a left bundle branch block (61). The presence of a left bundle branch block is associated with an asynchronous contraction pattern and worse outcome (62). In a large population of patients with
congestive heart failure, the presence of an left bundle branch block yielded a hazard ratio of 1.36 (95% confidence interval 1.15 to 1.61, p < 0.001) for all-cause mortality.

Recently, CRT has emerged as a new treatment strategy for this subgroup of heart failure patients (63). However, approximately one-third of the patients eligible for CRT do not have response to this therapy (64), which raised the demand for adequate patient selection.

As shown by Nelson et al. (65), mechanical dyssynchrony is a key predictor for CRT efficacy in these patients. They quantified circumferential myocardial strain using MR tissue tagging in 7 healthy subjects and 8 patients with DCM and conduction delay before CRT, and measured the change in dP/dt\text{max} during CRT. Circumferential shortening was significantly reduced in DCM hearts compared with control subjects ($-5.3 \pm 2.1\%$ vs. $-18.6 \pm 2.9\%$, $p < 0.001$), consistent with depressed myocardial function. In addition, a high variance in strain was observed in these patients ($201.4 \pm 84.3\%$ vs. $28.0 \pm 7.1\%$, $p < 0.001$), indicating a larger dispersion or heterogeneity of regional peak systolic strain. This indicator of mechanical dyssynchrony showed a good correlation with the change in dP/dt\text{max} during CRT ($r = 0.85$, $p < 0.008$).

A study by Zwanenburg et al. (66) using MR tissue tagging with high temporal resolution (14 ms) showed that in general the onset of circumferential shortening in DCM patients propagated from the septum to the lateral wall, which is opposite to the direction found in normal subjects. However, in patients with nonischemic DCM, this pattern of mechanical activation was quite uniform, whereas in ischemic DCM a wide range of directions of activation were found. As a consequence, the area with delayed activation may vary as well, and this may be one of the mechanical explanations of why CRT is less effective in ischemic DCM (66).

**Mechanical LV function after partial left ventriculectomy.**

Partial left ventriculectomy was developed as an alternative therapy for end-stage heart failure, but results were variable and good predictors of outcome were not available. In a study by Setser et al. (67), circumferential shortening was calculated at 3 short-axis levels in 24 DCM patients before partial left ventriculectomy and 3 and 12 months after surgery. Before surgery, the septum did not show shortening but was stretched during systole. At follow-up after surgery, LV mass and volume decreased ($p < 0.01$). Circumferential shortening especially in the septum improved significantly at both postsurgical time points. In addition, it was found that stretch (dyskinesia) of the basal septum before surgery increased the probability of event-free survival after 6 months. Left ventricular torsion was further reduced after ventriculotomy, probably as a result of normalization of wall stress, resulting in better myocardial perfusion and subsequently improved subendocardial contraction, counterbalancing the subepicardial contraction (68).

**Hypertrophic cardiomyopathy.** In general, global LV function is preserved in patients with asymmetric septal hypertrophic cardiomyopathy (HCM). However, strain analysis by CMR tissue tagging showed that the amount of regional myocardial contraction and the strain rate are both impaired in hypertrophied as well as in nonhypertrophied regions. In these patients, an asynchronous contraction pattern also is observed, indicated by a larger dispersion in time to peak strain for different regions (69). Compared with control subjects, the maximum circumferential shortening occurs earlier in systole in HCM patients, especially within the hypertrophied septum. The heterogeneity of regional myocardial mechanics in HCM patients may reflect the regional variation in the myocardial disarray and fibrosis that is characteristic of this disorder.

Probably as a result of the subendocardial depressed circumferential strain, LV torsion (counterclockwise rotation of the apex around the long axis relative to the base) was greater in HCM patients compared with control subjects ($19.9^\circ \pm 2.4^\circ$ versus $14.6^\circ \pm 2.7^\circ$, $p < 0.01$) as shown by Young et al. (70).

In patients with LV hypertrophy of causes other than a genetic disorder, diastolic function represented by strain rate is impaired and systolic function is preserved. A recently published study by Edvardsen et al. (34) showed in participants of the MESA (Multi-Ethnic Study of Atherosclerosis) study a direct relationship between regional diastolic dysfunction and increasing LV mass. Regional diastolic strain rate was significantly reduced in patients with hypertrophy ($1.5 \pm 1.1\, s^{-1}$) compared with subjects without hypertrophy ($2.2 \pm 1.1\, s^{-1}$, $p < 0.001$).

**Studies in valvular heart disease.** The chronic pressure overload in patients with aortic stenosis induces LV hypertrophy and may cause ventricular dilatation later in the disease process. As a consequence of severe hypertrophy, diastolic dysfunction is likely to occur. Coronary flow is reduced in hypertrophied ventricles as well, and more in subendocardial regions compared with subepicardial regions (37).

Importantly, decreased contractility and concomitant increased torsion as a result of hypoperfusion may occur far before irreversible tissue damage including increased collagen content and altered architecture is present (58–60). Therefore, decreased subendocardial contraction and/or increased torsion may serve as early indicators of (reversible) cardiac abnormalities in patients prone to the development of cardiac dysfunction.

Nagel et al. (71) and Stuber et al. (72) have shown that during systole a wringing motion is performed by the normal LV with clockwise rotation at the base ($-4.4^\circ \pm 1.6^\circ$) and counterclockwise rotation at the apex ($+6.8^\circ \pm 2.5^\circ$) when viewed from the apex. Untwisting occurs mainly during the isovolumetric relaxation period and is completed before diastolic filling starts.

In patients with aortic valve stenosis, systolic rotation at the base was reduced ($-2.9^\circ$ before aortic valve replacement [AVR] vs. $-4.2^\circ$ in control subjects), whereas apical rotation was increased ($22.2^\circ$ before AVR vs. $10.3^\circ$ in control
Maximum systolic torsion was increased in patients with aortic stenosis before valve replacement (25.1° ± 6.6° vs. 14.5° ± 3.7°). One year after valve replacement, torsion decreased to almost normal values, although basal rotation remained decreased.

**FUTURE DIRECTIONS**

The unique features provided by CMR tissue tagging allow for detailed evaluation of regional systolic and diastolic myocardial function in 3 dimensions. This is of great advantage for evaluation of patients with heart failure of different etiologies. As shown, myocardial strain and torsion are sensitive measures for subendocardial contraction and global untwisting (relaxation) of the heart under different pathophysiological conditions. Alterations in diastolic function are the very first sign in developing cardiomyopathy. Quantification of torsion, untwisting (rate), and diastolic strain rate with CMR tissue tagging opens the prospect of early detection of cardiac disease in patients prone to the development of cardiomyopathy.

Today, a major limitation remains the impossibility of routinely studying patients with a pacemaker and/or internal cardioverter-defibrillator. However, several individual cases and a limited number of animal and human studies have been published reporting on the effects and safety of magnetic resonance imaging and devices (74,75). Only under strict conditions, when special precautions are made regarding the functioning of the pacemaker, and when an appropriate imaging strategy is used, can a magnetic resonance imaging study be performed relatively safely in these patients (74). However, the risk of thermogenic damage and loss of capture remains present and cannot be overstated (76). In addition, the effects of magnetic resonance imaging on leads placed in the coronary sinus are currently unknown, and internal cardioverter-defibrillators are an absolute contraindication. Therefore, only when MR-compatible pacemaker systems become available can in vivo and follow-up studies be initiated, providing better insight in the relationship between electrical and mechanical activation.

**CONCLUSIONS**

As shown, CMR tissue tagging is an excellent noninvasive tool for studying myocardial function in detail with high spatial and temporal resolution. In contrast to other imaging modalities, it offers the possibility of quantifying regional, intramural myocardial function in 3 dimensions at every site of the heart, and provides detailed data not only on contraction but also on relaxation of the heart. In addition, CMR is capable of providing accurate data on global ventricular function geometry and tissue characteristics (presence of fibrosis) as well. Offering these unique features, CMR seems to be the noninvasive imaging modality of choice to fulfill the needs of fundamental science and study function, geometry, and tissue characteristics in relationship to impaired cardiac function and advanced therapies in vivo.

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