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iREVIEWS

STATE-OF-THE-ART PAPER

Left Ventricular Torsion

An Expanding Role in the Analysis of Myocardial Dysfunction

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During left ventricular (LV) torsion, the base rotates in an overall clockwise direction and the apex rotates in a counterclockwise direction when viewed from apex to base. LV torsion is followed by rapid untwisting, which contributes to ventricular filling. Because LV torsion is directly related to fiber orientation, it might depict subclinical abnormalities in heart function. Recently, ultrasound speckle tracking was introduced for quantification of LV torsion. This fast, widely available technique may contribute to a more rapid introduction of LV torsion as a clinical tool for detection of myocardial dysfunction. However, knowledge of the exact function and structure of the heart is fundamental for understanding the value of LV torsion. LV torsion has been investigated with different measurement methods during the past 2 decades, using cardiac magnetic resonance as the gold standard. The results obtained over the years are helpful for developing a standardized method to quantify LV torsion and have facilitated the interpretation and value of LV torsion before it can be used as a clinical tool. (J Am Coll Cardiol Img 2009;2:648–55) © 2009 by the American College of Cardiology Foundation

Torsion of the left ventricle (LV) is the wringing motion of the ventricle around its long axis induced by contracting myofibers in the LV wall (1). During initial isovolumic contraction, the apex and the base both rotate in a counterclockwise direction (2) when viewed from apex to base. Subsequently, during systole the base changes direction and starts to rotate in a clockwise direction, while the apex continues to rotate in counterclockwise direction (Fig. 1). LV torsion is followed by rapid isovolumic untwisting of the ventricle. During contraction, potential elastic energy is stored in the collagen matrix and cytoskeletal proteins (titin); its release (recoil) causes rapid untwisting (3,4) and contributes to active suction of blood from the atria (5).

The mode of contraction is determined by the oblique orientation of the myofiber sheets (6). Subendocardial fibers are right-hand-oriented; subepicardial fibers are left-handed. Therefore, LV torsion seems to occur predominantly in the direction of the subepicardial fibers. Because of its direct relation to fiber orientation, LV torsion is a valuable addition to strain measures such as longitudinal or circumferential shortening or radial thickening.

The first measurements on LV torsion were performed invasively (7–9). Because markers had to be implanted, measurements could only be performed in animals or patients who underwent cardiac surgery. Possibly, the invasive nature of these measurements also influenced LV torsion. During the last 2 decades, however, noninvasive imaging techniques became available to quantify LV torsion. Cardiac magnetic resonance (CMR) with tissue tagging

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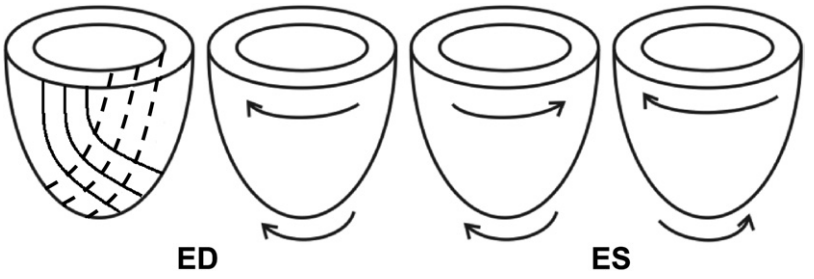

Figure 1. Schematic Drawing of LV Torsion

The **image on the left** shows the myofiber directions. **Solid lines** = epicardial region; **dashed lines** = endocardial region. The **image on the right** shows untwisting. ED = end-diastole; ES = end-systole; LV = left ventricle.

(10,11) was used as the gold standard, because this technique is able to create noninvasive markers over the entire myocardium in any anatomical plane that can be tracked throughout the cardiac cycle (Fig. 2). The recent introduction of speckle tracking in ultrasound again draws attention to LV torsion (12,13). The widespread availability of this tool may lead to a fast introduction of LV torsion as a clinical measure for detection of myocardial dysfunction. However, before LV torsion can be used as a clinical tool, the physiology of the torsional deformation should be well understood.

Methodological Issues and Physiological Fundamentals of LV Torsion

Over the years, torsional deformation of the LV has repeatedly been studied both in animals and in man. Different definitions of LV torsion were given in literature (Fig. 3). One definition describes LV torsion by the difference in rotation (φ) between base and apex (14), the twist. Another definition is the normalized twist, where this twist angle is divided by the distance (D) between the measured locations of base and apex (15). However, to make LV torsion comparable among differently sized hearts, the normalized twist should be multiplied by the mean radius (ρ) of base and apex (16):

$$T = \frac{(\phi_{apex} - \phi_{base}) \times (\rho_{apex} + \rho_{base})}{2D}$$

In this way, LV torsion (T) is directly related to the circumferential-longitudinal shear angle (Fig. 3). A unified method to calculate LV torsion should use this definition and must be independent of the measurement method. In this review, studies are interpreted with regard to this definition.

One of the first noninvasive, CMR-based LV torsion measurements in normal human volunteers was performed by Buchalter et al. (14). It was shown, that the twist angle increased with distance from base to apex. The circumferential-longitudinal shear angle, however, remained constant from base to apex and from endocardium to epicardium. Both twist and shear angles were counterclockwise as seen from the apex and greater in anterolateral regions than in posteroseptal regions.

Young et al. (17) stated that for calculation of LV material point rotation, the motion of the reference centroid during the cardiac cycle should be corrected for. This resulted in more similar rotations over different regions. A comparable approach was used by Lorenz et al. (2). Both studies demonstrate that the anterior and lateral walls show significantly higher twist angles than the septal and posterior walls. Subendocardial rotation was found to be higher than subepicardial rotation.

The larger anterolateral than inferoseptal rotation might be a result of misplacement of the reference centroid (18). Possibly, rotation occurs around the center of mass of the entire heart, which would be located more toward the inferoseptal region, instead of only around a centroid in the LV (Fig. 4).

The gradient in the twist angle over the long axis of the ventricle and the transmural differences in rotation seem to result in a constant circumferential-longitudinal shear angle over the LV. When such normalized measures of LV torsion are used, there even is a resemblance between murine and human LV torsion (19), despite a 10-fold difference in heart length, much shorter RR-interval and more than a 200-fold difference in ventricular mass. These results show that in mammals, LV torsion is

ABBREVIATIONS AND ACRONMYS

CAD = coronary artery disease
CMR = cardiac magnetic resonance
LV = left ventricle
MI = myocardial infarction

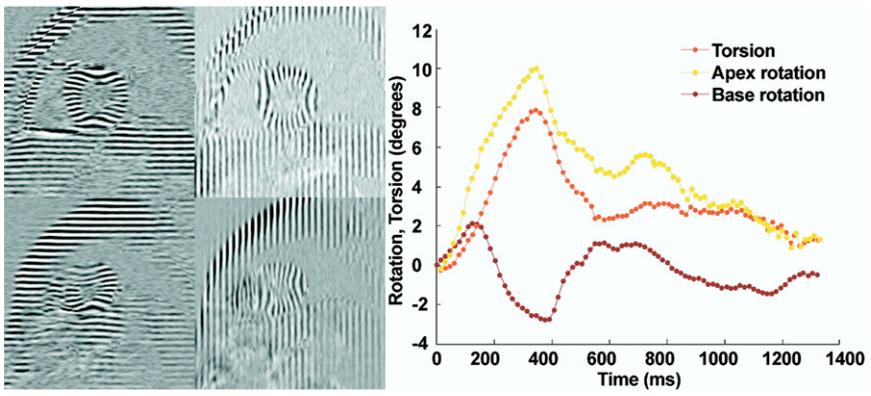

Figure 2. Example of Calculated Rotations and Torsion in a Healthy Subject Using CMR

(Left) Horizontally and vertically tagged end-systolic CMR images. Tag lines do not appear sharp, because sinusoidal tagging was used, which is optimal for harmonic phase tracking (47). **Top** images are basal, **bottom** images are apical. **(Right)** Corresponding rotation and torsion (circumferential-longitudinal shear angle) curves, starting at end-diastole and showing the entire cardiac cycle. However, the diastolic part of the curves is somewhat noisy, because of tag fading. This explains why the curves do not entirely return to 0. CMR = cardiac magnetic resonance.

fundamental to normal ventricular function. However, comparisons rely on the assumption that fiber orientations are similar; this was not specifically studied in the mouse heart.

Besides the magnitude of rotation, its timing also provides physiological information. A recent study measuring LV rotation in pigs (20) using ultrasound speckle tracking interestingly showed a significant difference in time-to-peak rotation between subendocardial and subepicardial myocardial layers. Unfortunately, no human data on this phenomenon is present yet.

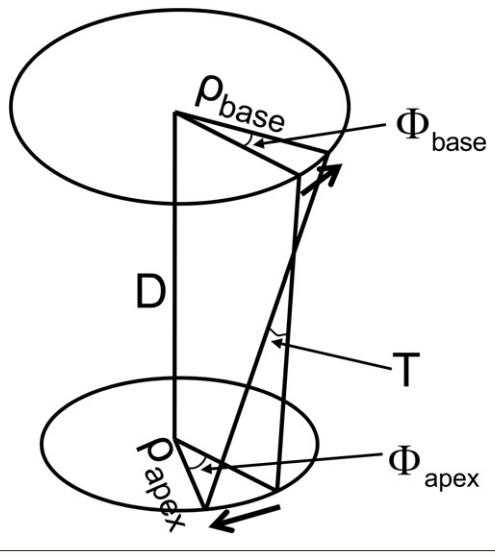

Figure 3. Different Definitions of LV Torsion

A sketch of a basal and an apical plane and the torsional deformation. Twist is defined as ($\Phi_{apex} - \Phi_{base}$), twist per unit length as ($\Phi_{apex} - \Phi_{base}$)/D, and left ventricle (LV) torsion T (circumferential-longitudinal shear angle) as ($\Phi_{apex} - \Phi_{base}$) ($\rho_{apex} - \rho_{base}$)/2D. Mostly, counterclockwise rotation as seen from the apex is positive.

Aelen et al. (16) investigated the relation between LV torsion and ejection in healthy volunteers. It had been calculated (21) that transmural fiber shortening is uniform when LV torsion is a function of the ratio of cavity volume to wall volume, which is related to the amount of circumferential shortening and wall thickening. The relation indeed exists in healthy volunteers and demonstrates that LV torsion is an important contributor to myocardial function. Dong et al. (22) found a similar relationship in the canine heart, where a positive relation between LV torsion and stroke volume and LV torsion and ejection fraction was found.

LV Torsion Under Specific Physiological Conditions

Inotropic and chronotropic stimulation. In a dog study by Rademakers et al. (3), it was found that maximum twist increased after inotropic stimulation by dobutamine infusion, and that untwisting was more rapid. This increase was confirmed by Buchalter et al. (23). To study chronotropic stimulation, in the latter study, the dogs were atrially paced, which also increased rotation. In a study by Sorger et al. (15), dogs were paced ventricularly (right ventricle apex and LV free wall). It was demonstrated that the clockwise rotation of the base and the counterclockwise rotation of the apex were preserved during pacing at all sites. However,

when compared with atrial pacing, ventricular pacing resulted in a reduction of normalized peak twist angles. Also, during untwisting, the ventricularly paced hearts exceeded the return-to-0 twist, resulting in a second small counterclockwise twist at end-diastole. The overshoot might have implications for LV filling, as LV pressure might be altered by the counterclockwise twist.

Both dobutamine and atrial pacing increase rotation, probably via the mechanism of the positive force-frequency relation. A human study by Notomi et al. (4) demonstrated that the LV twist angle, measured by tissue Doppler imaging, increased during exercise, which seems to be a similar effect. Furthermore, this study showed an accompanying exercise-induced increased velocity of untwisting, which suggests a link between systolic contraction and enhanced diastolic filling by active suction. In Figure 5, a chart is presented that proposes the effects of physiological exercise on torsion.

Ischemia. Buchalter et al. (23) also studied ischemia in the canine heart. Ischemia was induced at different locations. Anterior wall ischemia reduced rotation of only the anterior wall, but posterior wall ischemia reduced subepicardial posterior wall rotation and anterior wall rotation. Gibbons Kroeker et al. (24) showed that during coronary occlusion, peak apical rotation was delayed in the dog heart. Because regional ischemia influenced the (timing of) rotation of several other regions, the results suggest that LV rotation, and thereby LV torsion, depend on the complex fiber arrangement of the whole ventricle.

Load alterations. Gibbons Kroeker et al. (24,25) measured the effects of preload and afterload on apical rotation in open-chest dogs. With ischemia, maximum apex rotation occurred later. Decreasing preload and afterload (vena caval occlusion) resulted in an increase in amplitude of apex rotation, with earlier maximum rotation. Increasing preload (volume loading with saline) or afterload (single beat aortic occlusion) resulted in a small decrease and delay in maximum apex rotation. The results were confirmed in a study by MacGowan et al. (26).

The effects of preload and afterload on LV torsion in transplanted canine hearts were studied by Dong et al. (22). Increased preload (increased end-diastolic volume) caused an increase in twist angles. Twist angles decreased under increasing afterload (increased end-systolic volumes). Observed changes in torsion caused by changes in preload differ from what was found in the studies of Gibbons Kroeker et al. (24,25) and MacGowan et al. (26). A reason could be that in these studies only rotation was investigated, or that afterload was changed as well, because of an increase in end-systolic volume by the volume loading with saline. However, the observed changes in torsion due to changes in afterload were in agreement with these studies.

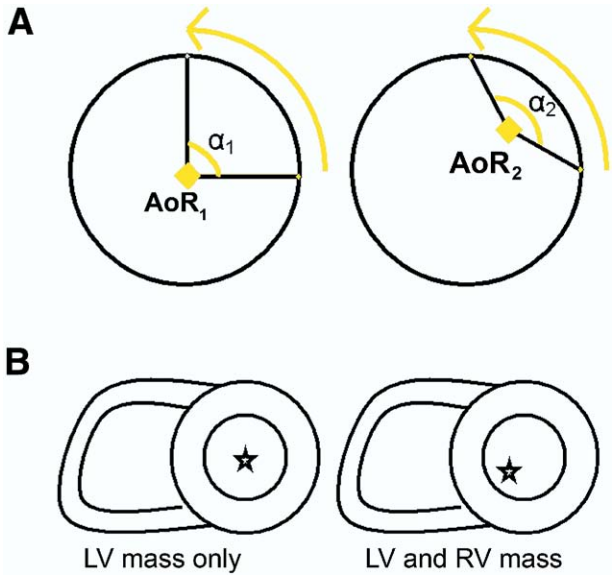

Figure 4. Effect of a Displaced AoR on Measured Rotation

A displaced AoR results in a different observed rotation angle for the same displacement **(A)**. Inclusion of the RV mass in the calculation of the AoR will move the AoR more toward inferoseptal **(B)**. Reprinted from Rüssel et al. (18), with permission from Biomed Central. AoR = axis of rotation; LV = left ventricle; RV = right ventricle.

Dong et al. (27) also hypothesized that the rate of untwisting might reflect the process of relaxation independent of left atrial pressure. As the extent of LV torsion is correlated with cavity pressure (22), the untwisting rate may be related to the rate of pressure fall. This untwisting rate was regressed against the relaxation time constant, which was obtained from hemodynamic analysis. Measurements were done under different loading and contractility circumstances. It was found that the untwisting rate correlated closely and reproducibly with the relaxation time constant, independently of pressure and load. Therefore, it is a parameter that can be used for the detailed study of diastolic function.

LV Torsion in Patients With Different Diseases

Pressure overload. Stuber et al. (28) studied LV torsion in pressure-overloaded hypertrophied hearts

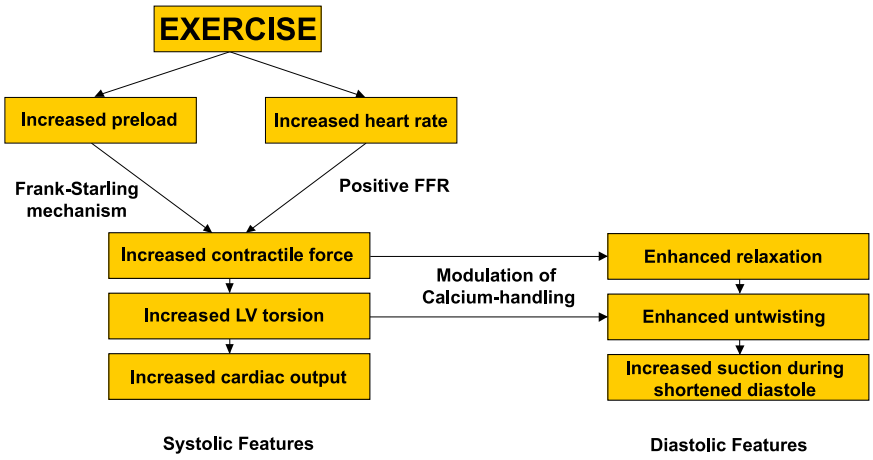

Figure 5. Pathways of Changes in LV Torsion During Physiological Exercise

Chart describing the pathways of changes in left ventricle (LV) torsion during physiological exercise. LV torsion and untwisting are interrelated. In order to increase cardiac input and output during exercise, increased and more rapid torsion and untwisting are required. FFR = force-frequency relation.

in patients with aortic stenosis, in athletes, and in a control group. They hypothesized that in athletes, wall stress would be normal due to the unchanged ratio of wall thickness to chamber radius. The normalized twist was not significantly different between athletes and controls. Whereas in patients with aortic stenosis, these values were significantly increased. The results found in this study confirm the relationships between LV torsion and pressure (22) and ejection (16). In a similar study (29), rotation and LV torsion were investigated in patients with aortic valve stenosis and in a control group. Twist angles were calculated at the point of maximum apex rotation. In patients, basal rotation was reduced, but apical rotation was increased and delayed. In addition, a delay in untwisting was observed during relaxation. A study by Sandstede et al. (30) also showed that patients with aortic valve stenosis have a significant increase of apical rotation, which is reduced after aortic valve replacement.

These changes in rotation are underlined by the parameter TransDif (31). When assuming myofiber shortening to be transmurally uniform, LV torsion to shortening ratio was predicted to be a fixed number, which was already confirmed (16). TransDif expresses the transmural uniformness of fiber shortening. In patients with aortic valve stenosis, TransDif was increased with respect to healthy subjects, suggesting impairment of subendocardial myocardial fiber shortening. In patients who were treated by aortic valve replacement, TransDif decreased but did not return entirely to normal.

Ischemic heart disease. In a study by Nagel et al. (32), cardiac rotation in patients with anterolateral myocardial infarction (MI) was investigated. In these patients, there was less systolic rotation at the apex and diastolic untwisting was delayed and prolonged in comparison with controls. The same was reported recently by Takeuchi et al. (33), who used speckle tracking echocardiography for LV torsion analysis in patients with anterior MI. Besides the effects of systolic dysfunction due to MI, which is reflected in less LV torsion, the subsequent impaired untwisting might reflect the occurrence of diastolic dysfunction in these patients.

Garot et al. (34) investigated systolic twist angles in patients after acute anterior MI and in a control group. Myocardial ischemia caused a decrease in LV twist angles in the patients with respect to the controls, which was related to global LV function. The same was observed by Buchalter et al. (23).

Paetsch et al. (35) investigated apical rotation with CMR tagging in patients suspected of coronary artery disease (CAD). Systolic rotational velocity was reduced in patients with CAD. Neither an increase in peak rotation nor an increase in systolic rotational velocity was found during low-dose dobutamine stress. Under high-dose dobutamine stress, these patients showed an increase in peak rotation and systolic rotation velocity. In patients without CAD, peak rotation and maximal rotation velocity (systolic and diastolic) increased with increasing dobutamine stress. In patients with CAD, time-to-peak untwist was delayed. Patients

could be identified with CAD from the diastolic parameter "time-to-peak untwist."

During the first seconds of ischemia, apical rotation was found to increase due to dysfunction of only the subendocardial fiber layer (24). The decrease in LV torsion during ongoing ischemia can be explained by the finding that reduced fiber shortening in the subepicardial fiber layers occurs as a result of impairment of mechanical function in the subendocardial layers, which is caused by tethering between the fiber layers via the stiff collagen network (36).

Cardiomyopathy. Setser et al. (37) studied LV torsion in patients suffering from dilated cardiomyopathy before and after partial left ventriculectomy. Clinical indices of cardiac function showed improvement, but twist angles remained unchanged, possibly due to disturbed fiber orientation after surgery.

MacGowan et al. (38) showed that in patients with dilated cardiomyopathy, endocardial circumferential-longitudinal shear was decreased relative to a control group, and epicardial shear was similar. Uniform transmural fiber shortening (although decreased) was maintained in these patients.

Reduced LV torsion in patients with dilated cardiomyopathy was found to be a predictor of response to cardiac resynchronization therapy and increased after 8 months of therapy (39).

In patients suffering from hypertrophic cardiomyopathy, LV torsion is increased with respect to a control group (40). The same holds for the velocity of untwisting (4).

Diabetes mellitus. Fonseca et al. (41) studied circumferential-longitudinal shear in type-2 diabetic patients with diastolic dysfunction and normal ejection fraction. Peak LV torsion and systolic torsion rate were greater in patients than in the control group. Peak rate of untwisting, however, did not differ between both groups, indicating impaired relaxation. Because peak circumferential and longitudinal strains were lower in the patient group, heart function seems to be compensated by increased LV torsion.

General heart failure. In a recent study (42) using speckle tracking echocardiography, the LV untwisting rate was studied. It was found that the LV untwisting rate is related to the peak twisting angle and the LV end-systolic volume, both in patients with decreased ejection fraction and in patients with diastolic dysfunction. This finding is in line with the studies on load alterations, which found decreased twist angles during increased end-systolic volumes.

Discussion and Perspectives

This review shows that LV torsion is essential for proper myocardial function. All findings show that LV torsion may be considered as a marker for cardiac disease. Additionally, quantification of LV torsion might be helpful in clinical decision making. It might indicate proper timing of aortic valve replacement or response to cardiac resynchronization therapy. Also, it could be used to monitor the effect of therapy.

Limited data is available on LV torsion during diastole yet, mainly because of the fading of CMR tags during the cardiac cycle. Newly developed CMR pulse sequences (43) and ultrasound speckle tracking are expected to solve this problem. Despite these limitations, it has been shown that there is a relationship between the LV pressure drop during isovolumic relaxation (recoiling), which generates diastolic suction, and untwisting (5,27,44).

Differences that were found between circumferential regions might be caused by imprecisely defined center points of the LV for the calculation of the rotation of the tissue (2,17). This effect, studied by Rüssel et al. (18), questions the added value of regional measurements and requires further investigation. Besides, LV torsion was found to be a more global measure of myocardial function (23). A standardized method for calculation of LV torsion should be adopted, to address this problem. Several reference values for LV torsion were presented in literature. However, different definitions of LV torsion were used. The LV torsion measurements should be comparable among different hearts. This can be achieved in the following ways.

LV torsion should be quantified as the circumferential-longitudinal shear angle (see Table 1 for reference values) to be clinically most useful. In this way, the length and radius of the heart are taken into account. Furthermore, circumferential-longitudinal shear is most related to the deformation process within the myocardial wall. Also, a unified way to describe LV torsion should be independent of the measurement method. Noninvasive measurement methods such as CMR tissue tagging and speckle tracking echocardiography must provide reproducible and comparable measurements of LV torsion, before they can be used as

Table 1. Reference Values for LV Torsion and Its Timing Between Base and Apex, Base and Mid, and Mid and Apex Levels in 12 Healthy Subjects, Calculated as the Circumferential-Longitudinal Shear Angle Using CMR Tagging

	Base-Apex	Base-Mid	Mid-Apex
Peak torsion, degrees	7.7 ± 1.4	8.2 ± 2.3	8.1 ± 1.1
Time-to-peak torsion, ms	366 ± 24	357 ± 33	370 ± 28

Adapted from Rüssel et al. (18).
LV = left ventricle; CMR = cardiac magnetic resonance.

clinical tools for detection of myocardial dysfunction. It might be difficult to incorporate the circumferential-longitudinal shear angle approach in echocardiography, due to the lack of a reference coordinate system. However, new methods such as 3-dimensional speckle tracking might be able to overcome this problem (45). Furthermore, recent ultrasound speckle tracking LV torsion studies are in agreement with CMR studies (12,13). The ultrasound speckle tracking method seems promising, also because of its excellent temporal resolution, but at this moment, CMR is still considered the gold standard for LV torsion measurements because of superior image quality and validated tissue tracking procedures (46,47).

The difference in magnitude of rotation between the endocardial and epicardial regions (circumferential-radial shear) is not in complete agreement with the net direction of rotation, which favors the subepicardial fibers. The larger subendocardial rotation might also be a mechanism to evenly distribute fiber shortening and circumferential-longitudinal shear over the transmurality of the myocardial wall.

To fully understand the physiological principles of LV torsion, additional studies are necessary. Despite important technical limitations, considerable knowledge about LV torsion in the healthy heart has already been obtained. However, only a uniform calculation method, which describes torsion as the circumferential-longitudinal shear angle over the complete cardiac cycle and corrects for centroid motion, will allow for the use of LV torsion as a measure for quantification of myocardial dysfunction, associated with a broad range of cardiac diseases. Because the amount and timing of LV torsion are directly related to the structure and function of the myocardium and myocytes, LV torsion is a promising measure for qualitative, as well as quantitative detection of (sub)clinical (systolic and diastolic) dysfunction.

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