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iREVIEWS

FROM PICTURES TO PRACTICE PARADIGMS

# Twist Mechanics of the Left Ventricle

## Principles and Application

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Left ventricular (LV) twist or torsion represents the mean longitudinal gradient of the net difference in clockwise and counterclockwise rotation of the LV apex and base, as viewed from LV apex. Twist during ejection predominantly deforms the subendocardial fiber matrix, resulting in storage of potential energy. Subsequent recoil of twist deformation is associated with the release of restoring forces, which contributes to LV diastolic relaxation and early diastolic filling. Noninvasive techniques such as magnetic resonance imaging and echocardiography are useful for understanding LV twist dynamics in clinical settings, and data regarding their relative merits and pitfalls are rapidly accumulating. This review is a focused update on the current and evolving applications of LV twist mechanics in clinical cardiology. First, the theoretical framework for understanding the physiological sequence of LV twist during a cardiac cycle is presented. Second, variations in LV twist encountered in different experimental and clinical situations are discussed. Finally, the review presents an algorithm for routine application of LV twist in clinical differentiation of patterns of LV dysfunction encountered in day-to-day practice. (J Am Coll Cardiol Img 2008;1:366–76)


Lower, in 1669, was the first to describe the twisting motion of the left ventricle (LV) as "... the wringing of a linen cloth to squeeze out the water" (1). Over the past 3 centuries, the twist deformation of the LV has continued to intrigue clinicians and researchers in their quest to understand the performance of human heart. Experimental and clinical explorations on LV twist have entailed use of numerous techniques such as implanted radiopaque markers (2), biplane cine angiography (3), sonomicrometry (4,5), optical devices (6), gyroscopic sensors (7), magnetic resonance imaging (MRI) (8–10), and echocardiography (11–14). Furthermore, growth of interest in the quantifying LV twist in clinical settings has resulted into development of innovative techniques in which LV twists are readily computed from grayscale cardiac ultrasound images obtained at the bedside.

### Uniform Definitions for Characterization of LV Twist Deformation

Terms such as LV rotation, twist, and torsion are often used interchangeably for explaining the wringing motion of the LV. For a uniform description, this review emphasizes that the term rotation be referred to the rotation of a short-axis sections of LV as viewed from the apical end and defined as the angle between radial lines connecting the center of mass of

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that specific cross-sectional plain to a specific point in the myocardial wall at end diastole and at any other time during systole (15). The unit of rotation is degrees or radians. The base and apex of the LV rotate in opposite directions. The terms twist and torsion refer to the same phenomenon and should be used for defining the base-to-apex gradient in the rotation angle along the longitudinal axis of the LV, expressed in degrees per centimeter or radians per meter (15). The absolute apex-to-base difference in LV rotation (also in degrees or radians) is stated as the net LV twist angle or the net LV torsion angle (15). Some investigators have also expressed torsion as the axial gradient in the rotation angle multiplied by the average of the outer radii in apical and basal cross-sectional planes, thereby representing the shear deformation angle on the epicardial surface (unit degrees or radians) (16). It must be emphasized that LV length and diameter change dynamically during a cardiac cycle, and therefore these normalization schemes permit comparison of only the peak magnitude of torsion for different sizes of LV (15,17).

## Temporal Sequence of LV Twist

Figure 1 shows the temporal sequence of LV twist. During isovolumic contraction, the LV apex shows brief clockwise rotation that reverses rapidly and becomes counterclockwise during LV ejection (18,19). The magnitude of peak rotation varies depending on the position of the cross-section viewed from the tip of LV apex. For standardization, therefore, the LV apical cross-section should be obtained well beyond the papillary muscle, with either none or the smallest view of the right ventricle (RV) in the cross-section. Counterclockwise twist in ejection is followed by untwisting (clockwise rotation) of the LV apex, which occurs during the isovolumic relaxation and the early period of diastole.

In contrast to the LV apex, rotation of the LV base is significantly lower in magnitude and opposite in direction. During isovolumic contraction, there is a brief counterclockwise rotation, which is followed by clockwise rotation during ejection and counterclockwise rotation during isovolumic relaxation and early diastolic filling.

Interestingly the description of LV apex rotation during the phase of isovolumic contraction has varied depending on the technique used. Studies that have measured LV apex rotation using cine-angiographic markers (18), sonomicrometry (11,14), rotational devices (6), and echocardiography (11,14) have recorded an initial clockwise motion of the LV apex and counterclockwise motion of the LV base during isovolumic contraction. However, studies with magnetic resonance tagging have reported that both the LV base and the apex rotate in a counterclockwise direction during isovolumic contraction (10,20). The reason for this discrepancy remains unclear and has been attributed to the lower temporal resolution of magnetic resonance tagging (14).

**ABBREVIATIONS AND ACRONYMS**

**LV** = left ventricle/ventricular
**MR** = mitral regurgitation
**MRI** = magnetic resonance imaging
**RV** = right ventricle/ventricular

## Link Between Myofiber Geometry and Twist Mechanics

In the LV myocardial wall, the myofibers geometry changes smoothly from a right-handed helix in the subendocardium to a left-handed helix in the subepicardium such that the helix angle varies continuously from positive at the endocardium to negative at the epicardium (Fig. 2). To provide a framework for interpreting LV twist, Taber et al. (21) proposed a model of helical layer architecture composed of obliquely aligned muscle fibers embedded in an isotropic matrix (Fig. 2A). As shown in the model, on one hand the contraction of the epicardial fibers

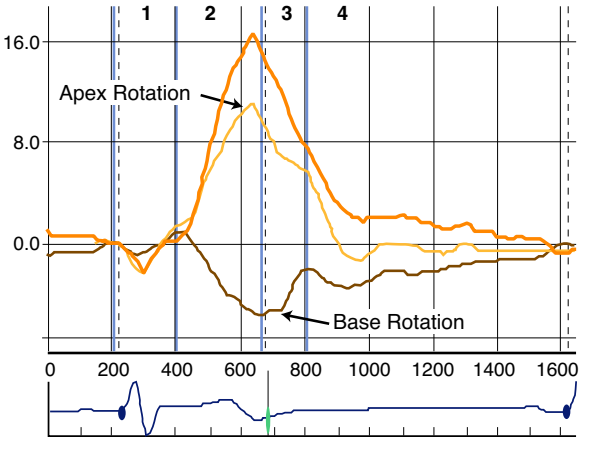


**Figure 1. Temporal Sequences of LV Twist During a Cardiac Cycle**

The left ventricular (LV) rotation from apical and basal cross-sections of LV has been obtained by speckle tracking of B-mode cardiac ultrasound images (GE Healthcare, Milwaukee, Wisconsin) in a normal healthy subject. The difference between the 2 rotations provides an estimate of net LV twist angle **(black line)**. During isovolumic contraction (phase 1), the apex shows a brief clockwise rotation and the base shows a brief counterclockwise rotation. During ejection (phase 2), the direction of rotation changes to counterclockwise at the LV apex and clockwise at the LV base, respectively. Torsional recoil occurs predominantly during the phase of isovolumic relaxation (phase 3) and early diastolic filling (phase 4).

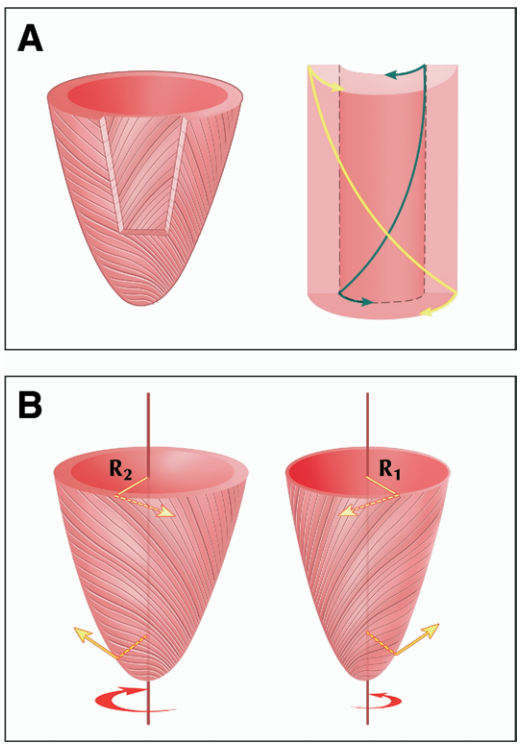


**Figure 2. Myofiber Architecture of LV and Models for Understanding LV Twist Dynamics**

Myofiber orientation in the left ventricular (LV) changes smoothly from a left-handed helix in the subepicardium to a right-handed helix in the subendocardium **(A, left)**. Thick-walled cylindrical myofibers models proposed by Ingels et al. **(A, right)** and Taber et al. **(B)**, showing the subendocardial fiber wrapped in a right-handed helix and a subepicardial fiber wrapped in a left-handed helix. **Arrows (A)** depict the circumferential components of force that results from force development in each fiber direction. The radii ($R_1$ for subendocardium and $R_2$ for the subepicardium) are the lever arms, which convert these circumferential components of force into torque about the long axis of the cylinder. The subepicardial fibers have a longer arm of moment than the subendocardial fibers ( $R_1 < R_2$). Figure illustration done by Rob Flewell.

will rotate the apex in counterclockwise and the base in clockwise direction, whereas on the other hand, contraction of subendocardial region will rotate the LV apex and base in exactly the opposite direction. When both layers contract simultaneously, a larger radius of rotation for the outer epicardial layer results in epicardial fibers having a mechanical advantage in dominating the overall direction of rotation (21).

With the onset of cardiac electromechanical activation, the subendocardial fibers near the mid and apical septal walls are first to be excited with an apex-to-base sequence of activation (Fig. 3) (22,23). Subendocardial shortening sequence is accompanied with subepicardial fiber stretching (24,25). Subendocardial shortening and subepicardial stretch both contribute to a brief clockwise rotation of LV apex (18,22,26,27). The transmural spread of electrical activation results in sequential subendocardial-to-subepicardial fiber shortening (24,25). Although the subendocardial forces exceed subepicardial forces, the larger radius of subepicardial region produces higher torque to dominate the direction of rotation. The large subepicardial torque is coupled transmurally to the midwall and subendocardium and results in global counterclockwise LV rotation near the apex and clockwise rotation near the LV base during ejection. In the subepicardium, this twist aids contraction in the principal fiber direction (18). In the midwall, this torque enhances shortening in the circumferential direction (18). In the subendocardium, this torque causes fiber rearrangement such that subendocardial fibers are sheared toward the LV cavity for LV wall thickening, whereas the LV base is pulled toward the apex, shortening the longitudinal axis of the LV. Twisting and shearing of the subendocardial fibers deforms the matrix and result in storage of potential energy, which is subsequently utilized for diastolic recoil (4).

The torsional recoil during isovolumic relaxation and early diastole releases the potential energy stored in the deformed matrix of the subendocardium (4,28,29). This process is facilitated by presence of lengthening-shortening gradients in the LV wall, which hasten lengthening of relaxed segments. For example, in experimental animal models, LV epicardium at the base is seen to lengthen while ongoing shortening occurs near the LV apex. However, the subendocardial fibers lengthen near the LV apex and recoil in clockwise direction, like a twisted coil that springs open, with ongoing shortening of the subendocardial region near LV base (22,23). The presence of simultaneous shortening and lengthening vectors of deformation within the LV wall allows diastolic restoration to be initiated without changes in LV volume.

Torsion helps bring a uniform distribution of LV fiber stress and fiber shortening across the wall (30). It has been shown in a mathematical model that normal torsion causes sarcomere shortening of 0.20 $\mu$m in the epicardium and 0.48 $\mu$m in the endocardium (31). However, elimination of the torsion would decreases epicardial shortening (0.10 $\mu$m), and increases endocardial shortening (0.55 $\mu$m). Thus the disappearance of torsion would increase endocardial stress and strain and, therefore, increase oxygen demand, thereby reducing the efficiency of LV systolic function.

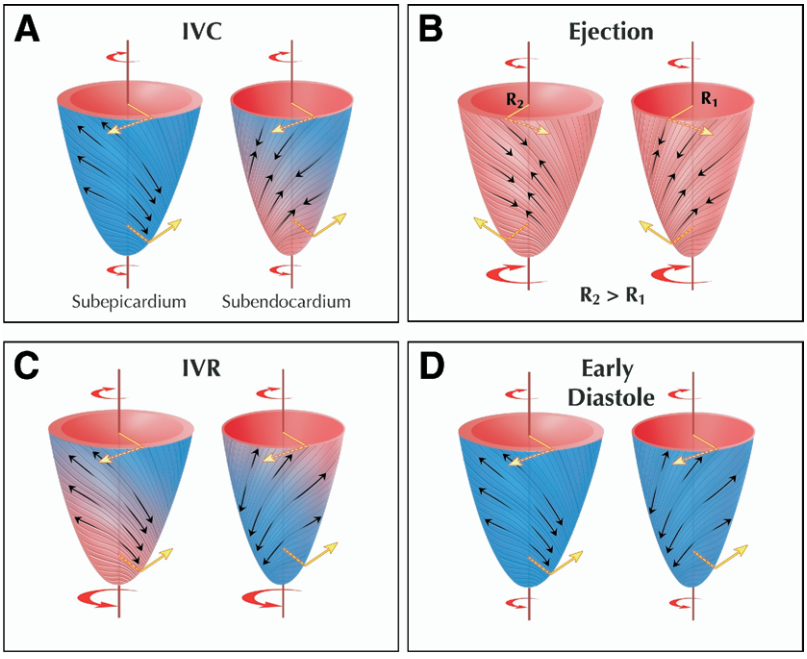


**Figure 3. Sequence of Twist Mechanics Explained in an Experimental Animal Model**

Electric and mechanical activation are initiated in the apical subendocardial region. During isovolumic contraction (IVC) **(A)**, the subendocardial myofibers (right-handed helix) shorten with stretching of the subepicardial myofibers (left-handed helix), producing a brief clockwise rotation of the apex and a counterclockwise rotation of the left ventricular base. During ejection **(B)**, the subendocardial and subepicardial layers shorten simultaneously, with shortening strains near the apex exceeding those of the base. The larger arm of moment of the subepicardial fibers dominates the direction of twist, causing rotation of the apex and base in counterclockwise and clockwise directions, respectively. During isovolumic relaxation (IVR) **(C)**, subepicardium lengthens from the base toward the apex and the subendocardium from the apex toward the base. The subsequent period of diastole is characterized by relaxation in both layers with minimum untwisting **(D)**. This figure refers to the experimental model that was used in Sengupta et al. (22). Figure illustration done by Rob Flewell.

## Physiological Variables Affecting LV Twist

The LV twists increase gradually from infancy to adulthood. Counterclockwise apical rotation is constant in its magnitude during childhood, whereas the basal rotation changes over age, initially counterclockwise in infancy to neutral in early childhood, and shows the adult clockwise pattern in adolescence (32). This progressive change has been attributed to the maturation of the helical myofiber architecture of the LV wall (32). Subsequently, with increasing age in adult life, subendocardial function may gradually attenuate, and LV twist increases further because of an unopposed increase in LV apical rotation (33,34). Age-related degenerative changes reduce the elastic resilience of the myocardial wall, and therefore the velocity of untwisting in early diastole progressively reduces (34).

Physiological variables such as preload, afterload, and contractility alter the extent of LV twist (Table 1) (35–37). Twist is greater with higher preload. For example, higher end-diastolic volumes of LV, with end-systolic volume held constant, produce higher LV twist. Similarly, afterload affects twist, that is, twist decreases at higher end-systolic volumes when end-diastolic volumes are held constant. The effect of preload on twist is about two-thirds as great as that of afterload. Like changes in loading conditions, increasing contractility increases LV twist; for example, positive inotropic interventions such as dobutamine infusion and paired pacing,

**Table 1. Physiological Variables Influencing Left Ventricular Twist Mechanics**

| Physiological Variables | Twist | $E_r$ |
|---|---|---|
| Increasing preload (35–37) | ↑ | ↓ |
| Increasing afterload (35–37) | ↓ | ↓* |
| Increasing contractility (9,36,38,39) | ↑ | ↑ |
| Exercise (40) | ↑ | ↑ |
| Increasing age (33,34) | ↑ | ↓* |

Numbers in parentheses correspond to the reference number in the References list. *Delayed onset of untwisting.
↓ = reduced; ↑ = increased; $E_r$ = early diastolic untwisting velocity.

greatly increase LV twist (9,36,38,39), whereas negative inotropic interventions markedly reduce twist (9).

In the intact circulation, changes in contractility are often accompanied by changes in loading conditions for increasing the twist mechanics of LV. For example, LV systolic twisting and untwisting can almost double with short-term exercise because of augmented rotation of both apical and basal levels (40), storing additional potential energy that is released for improving diastolic suction (41,42). Long-term exercise training may, however, reduce the LV twist at rest. Soccer players show lower LV twist values and untwisting velocities than nontrained individuals (43). It has been postulated that reduced LV twist in soccer players may represent increased torsional reserves that are used in increased-demand situations such as high-intensity sports. Indeed, a higher resting LV twist value, as seen with advancing age, is associated with attenuation of torsional reserves at peak exercise (44).

## Clinical Techniques for Measuring LV Rotation

The LV rotation can be measured in clinical practice noninvasively using MRI and echocardiography. For several years, MRI examinations were considered the reference standard for noninvasive assessment of cardiac biomechanics. The 2 most common MRI methods for measuring myocardial motion are tagging and phase contrast velocity mapping. Conventional analysis of tagged images entails computer-assisted detection of the epicardial border, endocardial border, and tag lines (45). Border and tag detection can be performed manually or semiautomatically; however, semiautomatic techniques generally require some extent of manual correction, and either technique is usually quite time consuming. Tissue phase mapping, on the other hand, directly encodes the velocity of myocardial motion into the MRI signal and offers high spatial resolution of the functional information (1 to 3 mm) (20). Because both methods in MRI are based on multiple breath-held 2-dimensional measurements, the temporal resolution is limited by the length of the breath-hold period to 30 to 80 ms. This limitation has been addressed by development of a respiratory-gated free-breathing method for tissue phase mapping that allows measurements with a temporal resolution comparable to tissue Doppler imaging. A major limitation remains the impossibility of routinely studying patients, particularly those with a pacemaker and/or an internal cardioverter-defibrillator.

Echocardiography has wide availability, and therefore is a more feasible technique for bedside assessment of LV twist, including for patients with a pacemaker and/or internal cardioverter-defibrillator. Applications for measuring twist using echocardiography were initially applied semiqualitatively by studying the rotational motion of papillary muscles (46). This was followed by attempts to decipher the rotational mechanics using tissue Doppler imaging (13); however, the angle dependency of Doppler imaging has remained a major limitation. Another echocardiographic method for motion estimation has gained recent acceptance and is based on 2-dimensional tracking of unique speckle patterns created by the constructive and destructive interference of ultrasound beams within tissue (11,12,14). These speckles are cross-correlated and tracked on a frame-by-frame basis. Because the tracking is fundamentally based on gray-scale B-mode images, it is independent of cardiac translation and angle dependency. The accuracy of speckle-tracking imaging has been validated against sonomicrometry and tagged MRI (12,14). However, the quality of tracking is dependent on the image quality and is vulnerable to dropouts of ultrasound data and reverberations. Moreover, clinical studies with speckle-tracking echocardiography have reported a wide variability in the values for resting LV systolic torsion (47). This may be related to the incongruent locations of LV apical and basal cross-sectional planes, errors related to through-plane motion, and the variable transmural depth of the region of interest for measuring LV rotation in each cross-sectional view. Methods for improving reproducibility of measurements should be addressed in future investigations.

## Clinical Applications

**Diastolic dysfunction.** Assessment of twist and peak untwisting rates were previously proposed to accurately reflect LV relaxation (48). Interestingly, however, recent studies indicate that LV twist may remain preserved in patients with diastolic dysfunction in presence of normal ejection fraction (49). For example, Wang et al. (49) studied 67 patients (34 with LV ejection fraction $<50\%$ and 33 with LV ejection fraction $>50\%$) undergoing simultaneous right heart catheterization and echocardiographic imaging and compared the LV twist mechanics with 20 healthy subjects as a control

group. The LV twisting and untwisting rates were reduced in patients with LV systolic dysfunction and depressed ejection fraction, but not in those with diastolic dysfunction and normal ejection fraction. The onset of untwisting occurred just before aortic valve closure in control subjects and was significantly delayed after aortic valve closure in patients with systolic and diastolic heart failure (Fig. 4). In another study of 49 patients with hypertension, twist was not different among groups of patients with or without LV hypertrophy, although early diastolic LV untwisting and untwisting rates were significantly delayed and reduced in parallel to the severity of LV hypertrophy, as assessed by LV mass index (50). Thus, LV twist is either preserved or augmented in patients with diastolic dysfunction and normal systolic performance. However, the onset of LV untwisting and the magnitude of peak untwisting velocities either remain normal or are reduced and significantly delayed (Table 2). More studies are required to explore the variability of this observation.

**Myocardial ischemia and the extent of LV infarct.** In patients with anterior wall myocardial infarction, peak circumferential strain in the apex is significantly depressed in patients with LV systolic dysfunction compared with those with preserved systolic function (51). In addition, LV twist is severely depressed in patients who have reduced LV ejection fraction, mainly because of the reduced magnitude of LV apical rotation. With the onset of systolic dysfunction, diastolic untwisting is also reduced and delayed (51). In contrast, systolic twist is maintained in patients with anterior wall myocardial infarction with relatively preserved LV systolic function (51,52). This is associated with a milder reduction of circumferential strain in the LV apex. The role of measuring LV twist during stress echocardiography has not been systematically evaluated. Because myocardial perfusion defects induced during stress testing are largely subendocar-

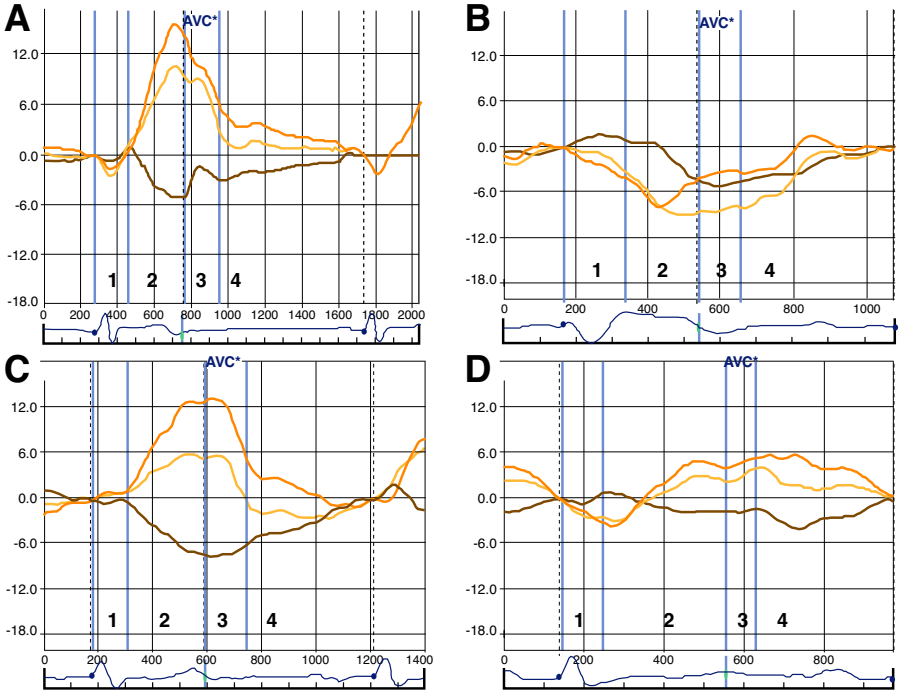


**Figure 4. LV Twist in Health and Disease**

Rotation of the left ventricular (LV) apex, the LV base, and the net LV twist angle (shown in **red**, **green**, and **black** colors, respectively) are assessed by speckle-tracking echocardiography in a normal subject **(A)**, a patient with dilated cardiomyopathy with systolic heart failure **(B)**, a patient with cardiac amyloidosis presenting as heart failure with normal ejection fraction (diastolic heart failure) **(C)**, and a patient with constrictive pericarditis **(D)**. Net ventricular twist is negative in dilated cardiomyopathy because of complete reversal of the LV apex rotation **(B)**. In contrast, a patient with amyloid cardiomyopathy shows relatively preserved magnitude of net LV twist angle. In a normal subject, the onset of untwisting occurs just before the aortic valve closure (AVC) **(A)**; however, in the patient with amyloid cardiomyopathy, the onset of untwisting is delayed after AVC **(C)**. The patient with constrictive pericarditis **(D)** shows reduced magnitude of net ventricular twist and marked delay in the onset of untwisting. Phases 1 through 4 are defined in the Figure 3 legend.

**Table 2. Left Ventricular Twist Mechanics in Different Cardiac Diseases**

| Cardiac Diseases | Twist | $E_r$ |
|---|---|---|
| Systolic heart failure | ↓ | ↓ |
| Diastolic heart failure (49,50) | N or ↑ | N or ↓ |
| Aortic stenosis (54–56) | N or ↑ | ↓ |
| Mitral regurgitation (58–60) | ↓ | ↓ |
| Transmural infarction (51) | ↓ | ↓ |
| Subendocardial ischemia | N | N or ↓ |
| Dilated cardiomyopathy (65) | ↓ | ↓ |
| Hypertrophic cardiomyopathy (70,71) | N or ↑ | ↓ |
| Restrictive cardiomyopathy (73) | N or ↑ | N |
| Constrictive pericarditis (73) | ↓ | ↓ |

Numbers in parentheses correspond to the reference number in the References list.
↓ = reduced; ↑ = increased; $E_r$ = early diastolic untwisting velocity; N = normal.

dial, it is likely that LV circumferential deformation and twist, which reflect subepicardial function, may remain unaltered. This postulate is supported by observations from experimental studies that have explored the influence of ischemia on LV rotation and reported greater-than-normal apical rotation with subendocardial ischemia and less-than-normal apical rotation with transmural ischemia (9,53).

**Transmural heterogeneity of LV mechanics in valvular heart diseases.** In aortic valve stenosis, coronary flow diminishes in the subendocardial region relative to the subepicardial region. The LV twist is significantly increased, although diastolic apical untwisting is prolonged in comparison with normal subjects (54–56). The delay in apical untwisting is associated with diastolic dysfunction and elevated LV end-diastolic filling pressures (54,55). After aortic valve replacement, LV twist normalizes. Level of recovery is, however, dependent on underlying coronary artery disease (57). Van der Toorn et al. (56) used the ratio of LV twist to LV shortening for assessing LV contractile function in patients with aortic stenosis. The twist shortening ratio was significantly higher in aortic stenosis than in a group of healthy volunteers. After aortic valve replacement, the ratio partially returned to normal. Similarly, subendocardial contractile function was found to be decreased before aortic valve replacement and significantly recovered 3 months after aortic valve replacement, even before LV remodeling was obvious (56).

Changes in LV twist have also been studied in patients with mitral regurgitation (MR) (58–60). In an experimental canine model, Tibayan et al. (58) showed that the progression from acute to chronic MR was associated with decreased peak systolic twist, delayed time to peak systolic twist (occurring after end-ejection in subjects with MR), and reduction in diastolic recoil. It has been suggested that chronic MR reduces systolic LV twist because of a decreased leverage of the epicardial fibers relative to the endocardial muscle fibers. Although increased preload will tend to increase systolic twist (35), chronic MR is associated with complex LV adaptive remodeling and eccentric hypertrophy. The effect of chronic MR on twist probably depends on the extent of subclinical LV systolic dysfunction. Peak untwisting velocity in MR remains normal, but correlates negatively with end-systolic dimension and regurgitant volume, suggesting that peak untwisting velocity, like peak systolic twist, depends on the stage of the disease (61).

**Congenital heart diseases.** There is limited information currently regarding the utility of assessing LV rotation in patients with congenital heart diseases. Because echocardiography represents the noninvasive tool most commonly used in pediatric cardiology, application of speckle-tracking echocardiography for bedside assessment of LV strain and twist deformation may provide important insights into mechanical adaptive responses of the RV and LV in congenital heart diseases. For example, in the normal heart, both RV and LV are coupled for twisting in the same direction (62). However, in patients with transposition of the great arteries, the morphologic RV supports the systemic circulation. It has been shown recently that the systemic RV contraction in these patients resembles that of the normal LV, however, without the ventricular twist (63). The global performance of the systemic ventricle is dependent more on the circumferential than the longitudinal free wall contraction, and may represent an adaptive response to the systemic load (64). As twist contributes to energy-efficient ejection, reduced twist might represent a potential for myocardial dysfunction (63). However, this hypothesis requires further prospective evaluation.

**LV twist variations in cardiomyopathy.** In dilated cardiomyopathy, amplitude of peak LV systolic twist is impaired in proportion to the global LV function (65). This reduction in LV twist is accounted for by marked attenuation of LV apical rotation, whereas basal rotation may be spared. In some cases, rotation of the apex may be abruptly interrupted such that in the initial part of systole, the LV base and apex rotate in the same direction (Fig. 4). After the initial part of the systole, the rotation diverges into 1 of 2 patterns: either continuation of identical

rotation at all levels for the remainder of systole (Fig. 4) or a divergence of rotation so that the apex and base rotate in opposite directions (66,67). There is limited information presently on the effects of therapeutic measures such as cardiac resynchronization therapy (68) or LV reduction surgeries (66,67) on LV twist. Preliminary data suggest that LV reduction surgery does not change LV twist, although the rate of early diastolic untwisting may improve (66,67). Similarly, LV torsional mechanics do not change after cardiac resynchronization therapy despite improvements in LV circumferential shortening mechanics (69). This suggests that loss of LV torsional mechanics in remodeled hearts may be difficult to restore once already established.

In contrast to dilated cardiomyopathy, patients with hypertrophic cardiomyopathy show relatively preserved net LV twist (70), although the apex-to-base progression of the LV twist sequence is altered. Rotation at the mid-LV level becomes clockwise, similar to the direction of rotation of the LV base (opposite to normal) (71). The area of null rotation, which represents the region of LV where rotation crosses over from clockwise into a counterclockwise direction, is apically displaced. This causes regional heterogeneity of LV twist, reducing the gradient of LV rotation for the basal aspect of LV, while exaggerating it toward the LV apex (71). During alcohol septal ablation, LV twist in hypertrophic cardiomyopathy may decrease transiently with occlusion of the septal perforator; however, after the injection of ethanol, twist increases to higher-than-baseline angles. Most of the improvement in twist is accounted for by an increase in LV apical rotation (72). Despite a preserved LV twist magnitude, patients with hypertrophic cardiomyopathy have reduced efficiency in generating untwisting. At rest, peak untwisting velocities are only marginally reduced in comparison with normal subjects. However, these differences became more dramatic with exercise, with the patients showing much lower untwisting velocities when compared with normal subjects (41).

**Differentiation of constrictive pericarditis from restrictive cardiomyopathy.** Both constrictive pericarditis and restrictive cardiomyopathy may have similar clinical presentations related to altered LV diastolic function. However, the mechanism leading to the LV diastolic function is different. In restrictive cardiomyopathy, the LV wall is resistant to stretch because of endocardial disease. On the other hand, tethering of subepicardial layers and an altered compliance of thickened pericardium is the main reason for LV diastolic filling impairment in constriction. The marked endocardial dysfunction with relative sparing of epicardial function leads to abnormal longitudinal mechanics with relative sparing of circumferential and twist mechanics in restrictive cardiomyopathy (73). However, in constrictive pericarditis, marked epicardial dysfunction leads to predominant impairment of circumferential shortening (74) and twist mechanics (73), while relatively sparing subendocardial longitudinal mechanics (Fig. 4). Similarly, congenital defects of pericardium cause a lack of LV twist while maintaining LV regional myocardial function (75), suggesting that normal pericardial layers may have important roles in modulating LV rotational mechanics.

## A Proposal for Assessing the Pattern of LV Dysfunction

Traditional concepts of heart failure have largely focused on the hemodynamic consequences of LV systolic dysfunction. Using a time-dependent model of heart failure, it has been proposed that diastolic and systolic heart failure are phenotypic expressions of the same disease process that evolves gradually as a continuum of clinical events (76). Assessment of cardiac muscle mechanics may provide superior pathophysiological insights into the mechanism of heart failure; however, this remains inadequately addressed.

The majority of progressive myocardial diseases, including coronary ischemia, tend to predominantly cause subendocardial dysfunction. This results in an early preferential involvement of longitudinal LV mechanics, which can be identified even in a subclinical state. The timing of contraction–relaxation cross-over is the most vulnerable period of myofibers mechanics (77,78). In early stages, therefore, ventricular relaxation either regionally or globally becomes abnormally slow and impaired with a progressive loss of the ventricle to modulate the timing of onset of relaxation. The epicardial function may remain relatively unaffected, and circumferential strain and twist either remains normal or shows exaggerated compensation for preserving the LV systolic performance. Compensatory features such as myocardial hypertrophy attempt to reduce subendocardial stress; however, such changes are usually maladaptive and detrimental (79). Furthermore, loss of cardiac muscle resilience also causes progressive delay in LV untwisting. Loss of early diastolic longitudinal relaxation and delayed un-

twisting attenuates LV diastolic performance, producing elevation of LV filling pressures. This stage manifests as heart failure with preserved systolic function. With further progression of disease, subepicardial function starts deteriorating, resulting in marked reduction of LV circumferential and twist mechanics. This causes progressive loss of LV ejection fraction and results in systolic heart failure. Acute transmural ischemia or infarction also results in simultaneous impairment of LV longitudinal and torsional mechanics, resulting in LV systolic dysfunction. Pericardial diseases, on the other hand, cause subepicardial tethering and predominant affection of LV torsional mechanics, while relatively sparing subendocardial function. A disease process such as radiation that affects both the pericardium and the subendocardial region may produce early attenuation of both longitudinal and circumferential LV function. Relative differences in longitudinal and torsional mechanics therefore may provide pathophysiological insight into the mechanism of LV dysfunction. Application of such algorithms requires prospective evaluation in future clinical trials.

## Final Comments

A growing body of evidence suggests that assessment of LV rotation and twist is feasible in clinical settings. The relationship between longitudinal and torsional mechanics of the LV provides insight into the transmural heterogeneity in myocardial contractile function. The presence of a subendocardial-to-subepicardial gradient in LV mechanics may provide a useful clinical measure for early recognition of a subclinical state that is likely to progress into either systolic or diastolic heart failure. With the advent of 3-dimensional echocardiography, newer algorithms for the characterization of ventricular twist mechanics hold promise for better understanding mechanisms of ventricular dysfunction and improving management of heart failure patients.

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