Left Ventricular Twist and Torsion
Research Observations and Clinical Applications

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Clinical Vignette

A 52-year-old white man visited his physician because he started experiencing shortness of breath on walking short distances at ground level. He had smoked half a packet of cigarettes daily for 40 years. Physical examination revealed a blood pressure of 147/95 mm Hg. Chest examination and chest x-ray were unremarkable, and ECG showed left atrial abnormality. The patient had normal serum electrolytes, blood sugar, and kidney function tests. A stress echocardiogram was ordered to exclude potential coronary artery disease. His resting echocardiography showed an ejection fraction (EF) of 60%, normal septal and posterior wall thickness, and mild diastolic dysfunction (septal early diastolic mitral annular velocity [e′] of 7 cm/s, early diastolic [E wave] to late diastolic [A wave] transmitral Doppler flow velocity ratio [E/A] of 1.4, E-wave deceleration time of 210 ms, E/e′ ratio of 9, and left atrial volume index of 44 mL/m^2; Figure 1A). There were no resting segmental wall motion abnormalities suggestive of ischemia. The patient exercised on a treadmill using Bruce protocol for 4 minutes and 43 s, and achieved 6.6 metabolic equivalent of task and maximum heart rate of 148 bpm (88% of his maximum age predicted heart rate). At peak exercise, the patient developed severe dyspnea and his blood pressure was 213/90 mm Hg. Post exercise echocardiography was acquired within 1 minute of exercise termination and showed EF of 69% and no segmental wall motion abnormalities, with Doppler recordings obtained at recovering heart rate of 125 bpm; showing a septal e′ velocity of 7.3 cm/s, E/A of 1.9, E-wave deceleration time of 110 ms, and E/e′ of 13.7, left atrial volume index of 35 mL/m^2 (Figure 1B). Ten minutes into the recovery period, the blood pressure returned to basal level (145/80 mm Hg). Compared with resting levels, the increased E/A ratio, shortened E-wave deceleration time and relatively increased E/e′ ratio suggested post exercise worsening of diastolic function with elevation of left ventricular (LV) filling pressures. To investigate the mechanistic basis of diastolic dysfunction in this patient, LV deformation was assessed offline using speckle-tracking echocardiography (STE). Besides characterizing the longitudinal and circumferential shortening, and radial thickening, the LV rotational deformation, that resembles the wringing of a towel, was also measured (Figures 2 and 3). This wringing deformation, also referred to as LV twist (LVT) and the subsequent recoil that occurs in diastole, referred to as untwist, were abnormal in this patient (Figure 4). At rest, the patient had mild diastolic dysfunction, which was associated with a higher than normal LVT and untwist values, compared with the published age-related normal values,[1,2] and low global longitudinal strain. At peak exercise, there was a significant worsening of the patient's diastolic parameters, which was associated with worsening untwist values and further reduction of global longitudinal strain, whereas LVT remained same in magnitude. The following sections provide an in-depth interpretation of these observations by revisiting the mechanistic basis of LVT, the pathophysiological factors affecting its magnitude, and the role of LVT abnormalities in the development of diastolic dysfunction in comparison with different phenotypic patterns of cardiac muscle dysfunction.

Basic Definitions

Twist, twist rate, untwist, and untwist rate (UTR) are commonly used terminologies for describing the features of systolic rotation and diastolic reverse rotation of the LV base and apex as viewed from the apex. Definitions of these terms are shown in Table 1. Importantly, LVT (expressed in degrees or radians) refers to the absolute difference in the magnitude of apical and basal rotation, and LV torsion (in degrees or radians per centimeter) refers to the normalized twist, where the twist angle is divided by the distance between the cross-sectional planes of the LV at the base and apex.

Temporal Sequence of LVT

Figure 2 shows the temporal sequence of LVT in isovolumic contraction, ejection, and isovolumic relaxation and early diastole. During isovolumic contraction, the LV exhibits brief untwist (clockwise rotation of the apex and counterclockwise rotation of the base), which is followed by twist during ejection (counterclockwise rotation of the apex and clockwise rotation of the base). This is followed by the recoil, that is, untwist (clockwise rotation of the apex and counterclockwise rotation of the base) that starts in isovolumic relaxation and continues into early diastole.[3] Untwisting parameters correlate with invasive indices of LV relaxation and suction (dp/dt and τ) but not with LV stiffness, suggesting that untwisting is a key mechanical event that aids LV early diastolic filling.[4]

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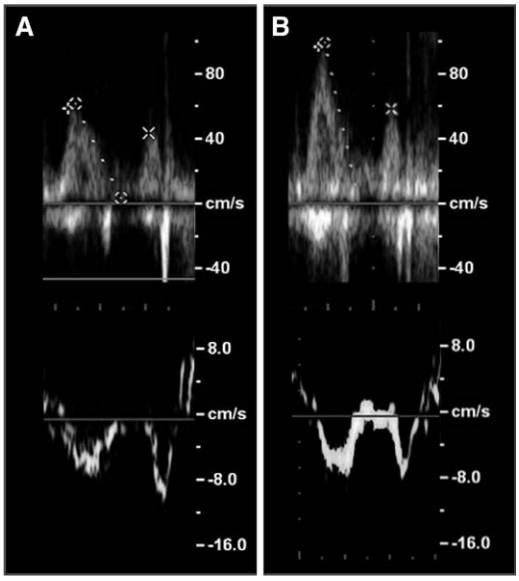

Figure 1. Doppler measurements of the patient in the clinical vignette. **Top**, The mitral flow pulsed Doppler early and late diastolic flow wave measurements. **Bottom**, Tissue Doppler measurements at the septal mitral annulus, (**A**) resting measurements, (**B**) postexercise measurements.

Importantly, in cases with abnormal LV relaxation, as tau increases, untwist is usually prolonged and the peak UTR is delayed.

Structural Basis of LVT

Myocardial fibers are a 3-dimensional (3D) continuum that change orientation gradually from a subendocradial right-handed helix to a subepicardial left-handed helix[3] (Figure 3A and 3B). This counter-directional helical arrangement of fibers also results in sliding or shear deformation.[5] Shear deformations occur in the circumferential-radial, longitudinal-radial, and circumferential-longitudinal planes. The largest shear deformation occurs in the circumferential-longitudinal plane and is visually identified as LVT.

Intuitively, the contraction of the subepicardial fibers would rotate the apex counterclockwise and the base clockwise, whereas contraction of subendocardial fibers would rotate the apex and base in exactly the opposite directions (Figure 3C). The reason these opposing forces do not result in nulling of LV rotation is because of the greater rotational radius of the outer epicardial layer, which exerts a larger lever arm force and, therefore, dominates the overall direction of rotation. However, myocardial electric activation is not transmurally homogenous. Myofiber shortening occurs earlier in the endocardium; therefore, subendocardial shortening during isovolumic contraction is associated with subepicardial stretch that causes a brief LV untwist. During the ejection period, transmural contraction produces higher subepicardial torque, which dominates the direction of rotation over the subendocardial fibers. The torque produced by the subepicardial twist arranges the subendocardial fibers such that they are sheared toward the LV cavity causing enhanced radial LV wall thickening.[3] Myocardial shear stores energy from systole into the deformed myocardial matrix. With the onset of relaxation, the stored energy is released much like a spring uncoiling under shape memory, facilitating rapid diastolic recoil, and suction.

Noninvasive Imaging for Assessment of LVT Mechanics

LVT mechanics can be assessed noninvasively using echocardiography[6] and cardiac magnetic resonance (CMR).[7] Echocardiographic assessment is feasible using color tissue Doppler and

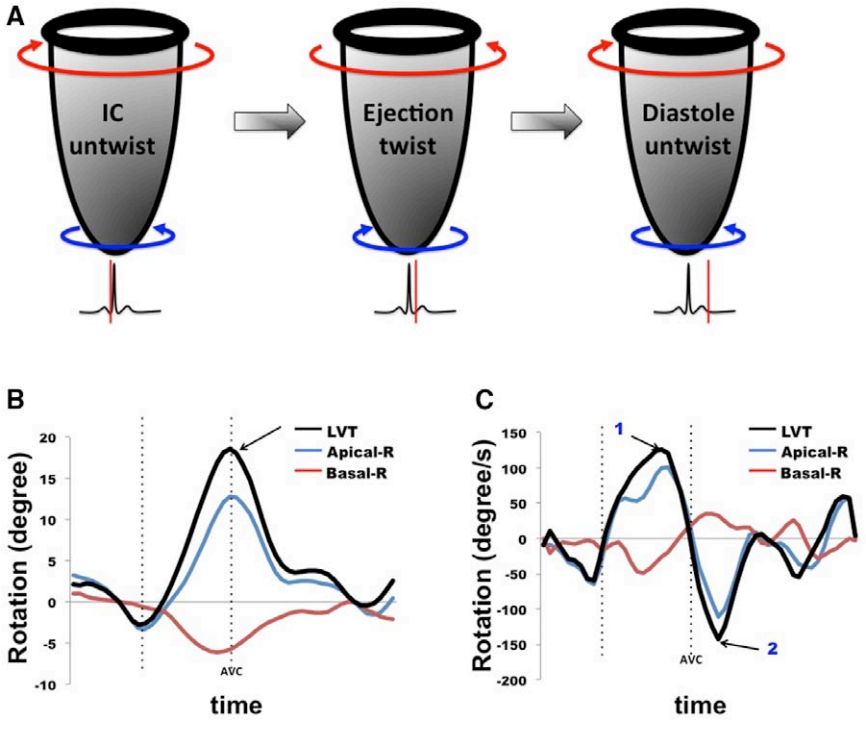

Figure 2. Temporal Sequence of left ventricular twist (LVT). **A**, Relative rotation of LV base (red curved arrow), and apex (blue curved arrow) during isovolumic contraction, ejection, and isovolumic relaxation and early diastole. **B**, LV rotational curves for the LV base (red line) and apex (blue line) from a normal healthy subject. LVT (black line and arrow) is the peak systolic difference between basal and apical rotation (arrow). **C**, LV rotational rate curves for LV base (red line) and apex (blue line) were derived from the same healthy subject in (**B**). LVT rate (black line, arrow 1) is measured as the peak systolic difference of basal and apical rotational rates, and LV untwist rate (black line, arrow 2) is the peak early diastolic difference between apical and basal reverse rotations.

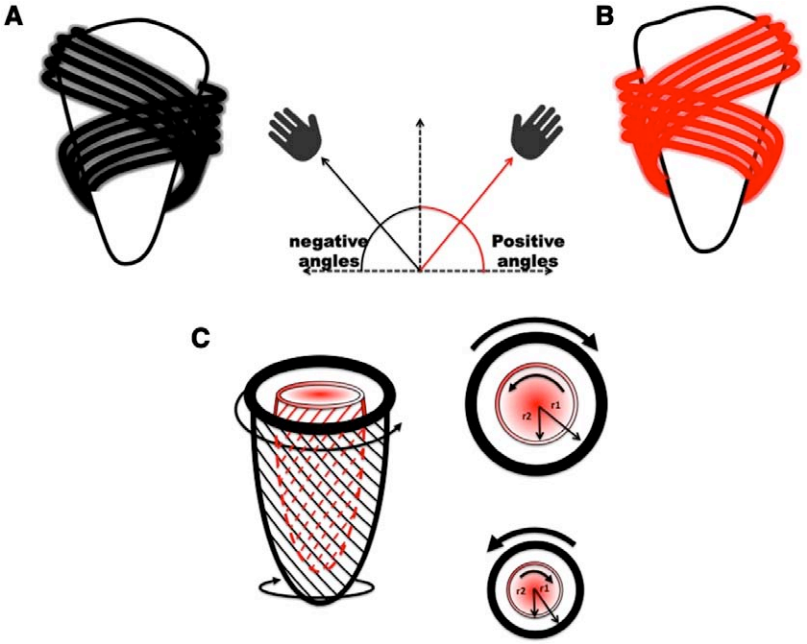

Figure 3. Myocardial fiber architecture. **A**, Subepicardial fibers wrapped around the left ventricle (LV) in a left-handed helix. **B**, Subendocardial fibers wrapped around the LV in a right-handed helix. **C**, The outer epicardial layer (in black) rotates the base in a clockwise direction and the apex in a counterclockwise direction, whereas the inner endocardial layer (in red) rotates the apex and base in exactly opposite directions. Because of the larger epicardial radius (r1) and the smaller endocardial radius (r2), the epicardial rotation dominates the overall LV rotational direction. AVC indicates aortic valve closure; IC, isovolumic contraction; and LVT, LV twist.

speckle-tracking imaging (STE). CMR assessment techniques include tissue tagging and phase contrast velocity mapping.

Speckle-Tracking Echocardiography

In the above clinical vignette, 2D-STE was used for assessment of LVT mechanics. 2D-STE has especially gained wide publicity, being relatively angle independent and widely available at the bedside.[6] For the assessment of LV rotational mechanics, images are obtained from the short axis of the LV base at the mitral valve level, and apex (Figure 5). The accuracy of 2D-STE has been validated against sonomicrometry and tagged CMR.[6] However, accurate tracking is dependent on the image quality and has interobserver variability. This may be related to the variable selection of LV apical and basal planes, through-plane motion, and the variable transmural depth of the region of interest. Because most of the inaccuracies result from inappropriate selection of the apical plane, attention should be focused on obtaining LV apical cross section well beyond the papillary muscle, with none or smallest view of the right ventricle. Three-dimensional (3D)-STE has also been recently used to calculate LV rotational and twist mechanics. The thicker sector of 3D-STE allows for capturing as many speckles as possible and tracking them in all directions while overcoming through-plane motion that usually affects 2D-STE, which allows for measurements at lower frame rates. Results are, however, variable because of the limited spatiotemporal resolution of current 3D-echocardiography systems,[8] which decreases its ability in capturing events occurring in fast phases of the cardiac cycle, such as isovolumic contraction and isovolumic relaxation.

Cardiac Magnetic Resonance

For several years, CMR was considered the reference standard for noninvasive assessment of cardiac biomechanics. The 2 most common CMR methods for measuring myocardial motion are tissue tagging and phase contrast velocity mapping. Tagged image analysis can be performed manually or semiautomatically, both of which, however, can be relatively time consuming. Tissue phase mapping, however, directly encodes myocardial motion velocity into the CMR signal and offers high spatial resolution. The need for multiple breath-holding in both methods, however, significantly affects the temporal resolution. This limitation has been addressed by the development of a respiratory-gated free-breathing tissue phase mapping, allowing measurements with a temporal resolution comparable to tissue Doppler imaging.[9] The contraindication in patients with pacemakers or internal cardioverter–defibrillator remains a major limitation of all CMR techniques.

Physiological Determinants of LVT

Age

LVT magnitudes are lower in values during infancy and increase progressively until adulthood (Table 2).[1,2] At infancy, both LV apex and base exhibit a counterclockwise rotation during systole. Basal rotation changes direction to become neutral during adolescence and then clockwise in adulthood.[10] This progressive change has been attributed to the maturation of the LV helical myofiber architecture.[10] In adults, regardless of sex, LVT continues to increase in magnitude, whereas untwist attenuates, and delays. The underlying mechanism seems to be a gradual subendocardial dysfunction associated with the aging process.[11] In the case presented above, age therefore might be a factor responsible for higher than normal resting LVT.

Changes of Loading and Contractility

In normal hearts, LVT mechanics are load-dependent (table 2). With afterload held constant, increasing preload (eg, by saline infusion and in pregnancy) augments LVT and untwist [12,13]; and with preload held constant, increasing afterload, for example, isometric handgrip, decreases LVT and untwist [14], whereas decreasing afterload, for example, using

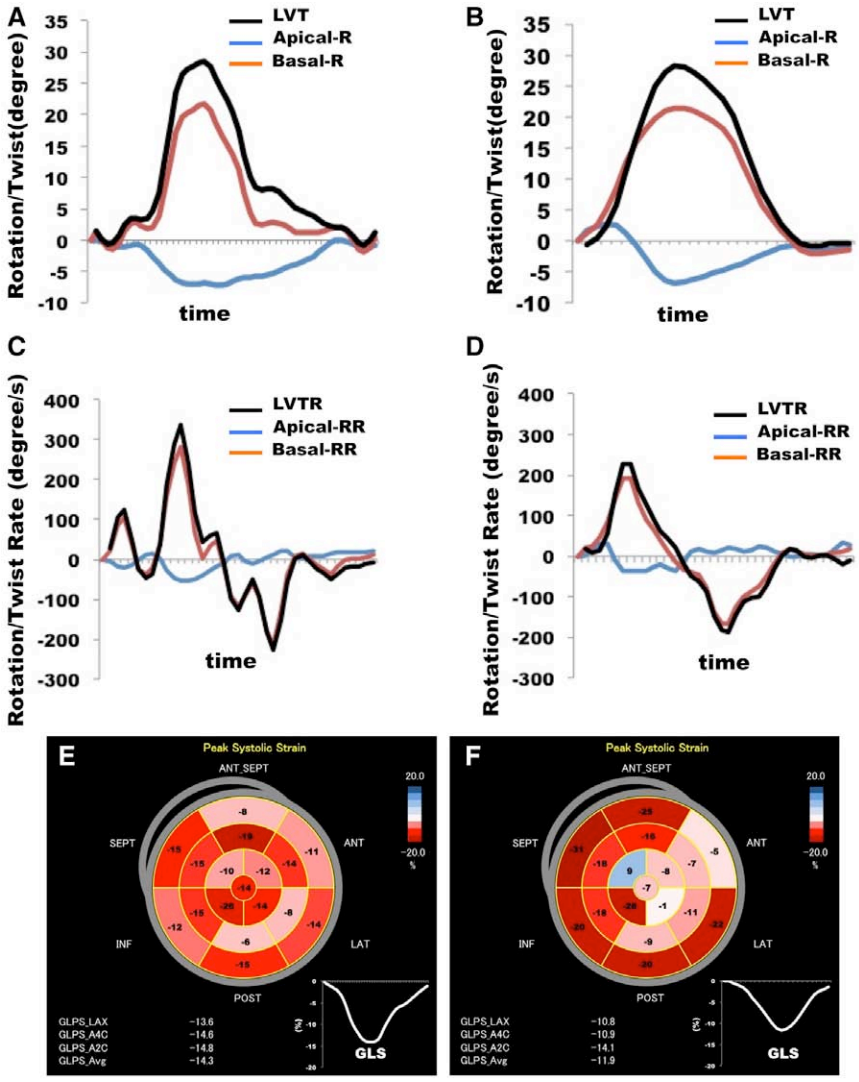

Figure 4. Left ventrivular twist (LVT) parameters obtained from 2-dimensional speckle-tracking echocardiography at rest and at peak exercise of the patient mentioned in the clinical vignette. **A**, Rotational curves at rest showing preserved LVT values. **B**, Rotational curves at peak exercise showing that LVT values failed to increase (blunted exercise reserve). **C**, LV rotational rate curves at rest showing preserved systolic and diastolic untwist rates (UTRs). **D**, LV rotational rate curves at peak exercise showing that LVT rate and UTR decreased compared with resting values. **E**, Polar map and curve for global longitudinal strain (GLS) at rest showed lower than normal values. **F**, GLS was further worsened at peak exercise.

arterial vasodilators such as sodium nitroprosside, increases LVT and untwist [15]. In dilated globular hearts, however, LVT becomes load independent.[16] In the patient presented above, the developed severe systolic hypertension during exercise increased LV afterload, which may have contributed to the twist–untwist abnormalities observed. Changes in contractility also affect LVT mechanics. Positive inotropic interventions, for example, dobutamine infusion and paired pacing, increase LVT,[17] whereas negative inotropic interventions markedly reduce LVT.[17]

Effect of Exercise

Exercise is also associated with increased contractility, thus, under normal conditions, is associated with a significant increase in both LVT and untwist (Table 2). Importantly, young healthy individuals possess twist reserve mechanisms, by which LVT increases significantly at incremental exercise loads.[18] Exercise twist reserve is a mechanism by which the heart increases its pumping abilities to be able to meet the increasing metabolic needs with exercise. Long-term physical training reduces resting LVT and untwist, thereby increasing the range of exercise twist reserve mechanism.[19] The type of training has varying effects on LVT. Endurance training (eg, marathon runners) increases all resting myocardial mechanics, including apical rotational velocity, LVT, and untwist with reduced age-related effects on twist–untwist mechanics.[20] Strength training (eg, weight lifters), however, is not known to be associated with these differences. Exercise-related twist reserve becomes blunted by age and in the presence of myocardial dysfunction. In the case presented above, the inability to increase LVT with exercise suggests the loss of exercise twist reserve, which was further associated with the blunted untwist at peak exercise.

Impact of Myocardial Structural Abnormalities on LVT

Disruption of normal myocardial geometry greatly affects LVT mechanics. In a hypertensive patient, the relative increase in wall thickness produces larger radial differences between the endocardium and the epicardium that increases subepicardial fiber leverage, resulting in the augmentation of LVT.[21] The control of blood pressure, however, is associated

Table 1. Basic Definitions and Parameters Used to Assess LV Twist Mechanics

Parameters	Definition
Systolic	
Apical rotation (°)	Peak counterclockwise systolic rotation of the LV apical short-axis cross section as viewed from the apex
Apical rotation rate (°/s)	Peak velocity of apical counterclockwise rotation
Basal rotation (°)	Peak clockwise systolic rotation of the LV basal short-axis cross section level as viewed from the apex
Basal rotation rate (°/s)	Peak velocity of basal clockwise rotation
LV twist (°)	Peak difference in systolic rotations of LV apex and base as viewed from the apex
LV torsion (°/cm)	Normalized twist: twist angle divided by the distance between the measured locations of base and apex
LV twist rate (°/s)	Peak velocity of LVT
Diastolic	
Apical reverse rotation (°)	Peak clockwise diastolic reverse rotation of the LV apical short-axis cross section as viewed from the apex
Apical reverse rotation rate (°/s)	Peak velocity of apical diastolic reverse rotation
Basal reverse rotation (°)	Peak counterclockwise diastolic reverse rotation of the LV basal short-axis cross section as viewed from the apex
Basal reverse rotation (°/s)	Peak velocity of basal diastolic reverse rotation
LV untwist (°)	Difference in diastolic reverse rotations of LV apex and base as viewed from the apex, measured as percentage of untwist from aortic valve closure to mitral valve opening (% UT in IVR)
Untwist rate (°/s)	Peak velocity of UT

IVR indicates isovolumic relaxation; LV, left ventricle; LVT, LV twist; and UT, untwist.

with normalization of the increased LVT.[22] Indeed the regression of the hypertrophied LV is associated with apoptosis at the subepicardial level, which normalizes the relationship between the subepicardial and subendocardial fibers.[23]

Changes in myocardial fiber architecture may also be seen in other conditions. For example, in congenital anomalies, such as situs inversus totalis, the orientation of the apical and epicardial basal fibers is normal, but the orientation of the deeper endocardial basal fibers is inverted.[24] Twist is, thus, normal at the apex, but changes direction at the base,[25,26] causing absence of relative apex to base rotation.[26] Similarly, in patients with LV noncompaction and hypoplastic hearts, the absence of normal fiber architecture causes both the LV base and apex to rotate in the same direction, exhibiting none or minimal twist, a physical type of rotation that is called rigid body rotation.[27]

Acquired disorders associated with fibrotic changes and scar formation may also change cardiac fiber geometry. For example, after myocardial infarction, the infarcted subendocardium remodels with scar formation and shrinkage, whereas the subepicardium shows compensatory hypertrophy resulting in preservation of LVT, which compensates for the reduced radial and longitudinal mechanics.[28] A large transmural infarction is associated with sufficient scarring at the subepicardial level and, therefore, associated with reduction of LVT.[29]

Impact of Myocardial Contractile Dysfunction on LVT

Changes in LVT mechanics can occur in the absence of macroscopic structural abnormalities. In the presence of long-standing cardiovascular risk factors such as hypertension,[22] diabetes mellitus,[30] obesity, and hyperlipidemia,[31] microscopic interstitial matrix changes as a result of increased collagen degradation products,[32] intramyocardial fibrosis,[33] or development of microvascular ischemia,[31] cause LV dysfunction at the subendocardial level. As long as systolic function remains preserved, the resultant subendocardial dysfunction will reduce LV mechanics in the longitudinal direction, whereas LVT remains preserved or even increases as a result of the unopposed contraction of the subepicardial fiber. As such, LVT might serve as a compensatory mechanism, by which LV systolic function is preserved, despite the blunted longitudinal mechanics. However, untwist is always reduced and delayed in these patients, which explains progression of diastolic dysfunction. It is important to mention that UTR, in addition to being related to LV relaxation, is also affected by LV restoring forces and loading conditions. Because restoring forces are generated by systolic contraction, changes in UTR may occur because of changes in systolic function rather than changes in LV relaxation alone.[34] As such, changes in UTR might not be an accurate representation of the diastolic dysfunction in patients with heart failure with preserved ejection fraction. This might explain the increased resting UTR in the above presented case, despite seemingly having impaired LV relaxation.

Furthermore, the reduction of LVEF is associated with reduction in LVT and untwist, signifying a more advanced stage of the myocardial dysfunction and exhaustion of LVT compensatory mechanisms. The intricate relationship of LVT with the extent of transmural myocardial function and compensatory mechanism may partly explain the phenotypic presentations in patients with heart failure with preserved or reduced EF (Figure 5; Table 3).

LVT Mechanics in Specific Cardiac Pathologies

Cardiomyopathy

Changes in LVT are closely related to the changes observed in global LV remodeling and reduction in EF in patients with cardiomyopathy. Although LVT is impaired in patients with reduced EF, it may be better preserved in those with normal or increased EF.[18] All cardiomyopathy phenotypes, however, show abnormal untwist values and blunted exercise reserve[18] (Table 4). In dilated cardiomyopathy, twist mechanics are blunted because of the increased LV sphericity, which leads

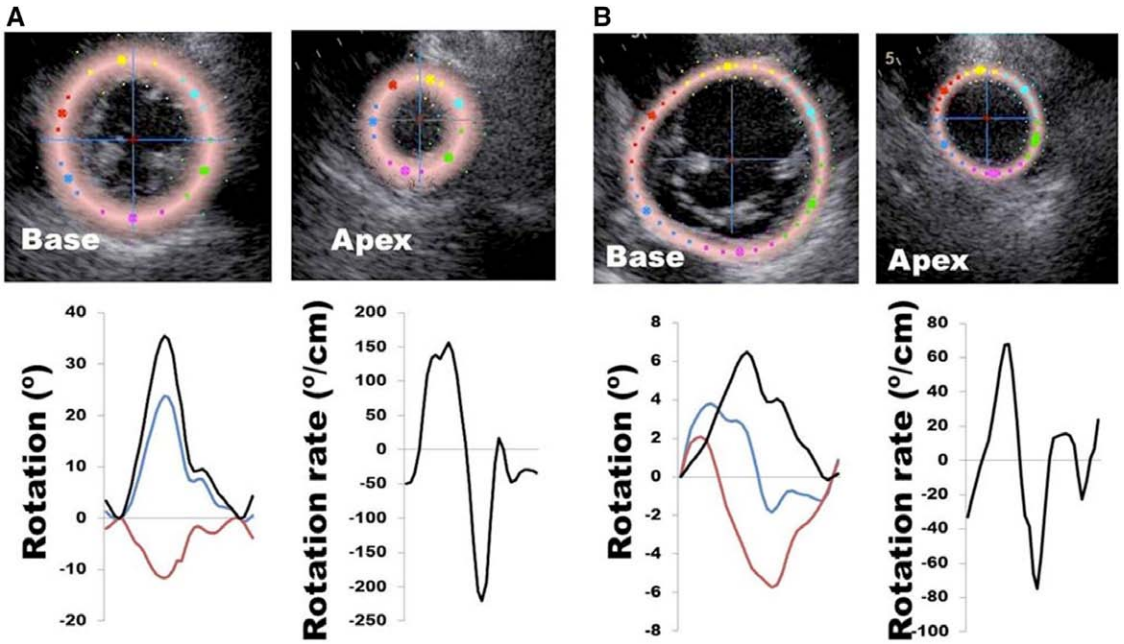

Figure 5. Examples of left ventricular (LV) rotational mechanics in a patient with heart failure with preserved ejection fraction (HFPEF) secondary to LV hypertrophy (**A**), and a patient with heart failure with reduced EF (HFREF) secondary to dilated cardiomyopathy (**B**). In HFPEF, all rotational values were higher than normal, serving as a compensatory mechanism for preservation of normal EF. In HFREF, all rotational values were decreased signifying exhaustion of LV twist reserve mechanisms, contributing to the reduced EF. Blue lines represent apical rotation, and red lines represent basal rotation.

to widening of the apex and loss of the oblique architecture of the apical fibers.[35] Twist mechanics are also affected by interventions. For example, LVT improves in responders to cardiac resynchronization therapy and is predictive of post cardiac resynchronization therapy reverse remodeling.[36] In patients treated by cardiac transplantation, LVT is reduced in biopsy-proven rejection.[37]

In hypertrophic cardiomyopathy, the relative increase in wall thickness produces larger radial differences between the endocardium and the epicardium resulting in augmentation of peak LVT.[38] The writhing area (ie, area of null rotation)[3] is displaced apically in hypertrophic cardiomyopathy because of myofiber disarray.[18] Interestingly, LVT and untwist are found to increase in genotypically positive hypertrophic cardiomyopathy carriers, despite having normal LV wall thickness.[39] The development of worsening LV outflow obstruction increases the afterload and may reduce LVT and untwist.[40] Surgical septal myomectomy has been reported to normalize these abnormalities.[41]

Table 2. Effect of Physiological Variables on Left Ventricular Twist Mechanics in Comparison to Resting Healthy Young Controls

	Systolic Twist	Untwist Rate
Infancy	↓	↓
Aging	↑	↓
Increased preload	↑	↑
Increased afterload	↓	↓
Arterial vasodilation	↑	↑
Positive inotropics	↑	↑
Isometric exercise	↓	↓
Exercise	↑	↑
Pregnancy	↑	↑

Constrictive Pericarditis Versus Restrictive Cardiomyopathy

Similar clinical presentations can be found in both constrictive pericarditis and restrictive cardiomyopathy because of the development of diastolic dysfunction, although with different mechanisms. Restrictive cardiomyopathy can be caused by infiltrative diseases, such as the amyloid heart disease. In amyloid restrictive cardiomyopathy, there is marked endocardial dysfunction, with relatively normal epicardial function, causing abnormal longitudinal mechanics and relatively normal circumferential and twist mechanics[42] (Table 4). However, in constrictive pericarditis, the marked epicardial dysfunction leads to impairment of circumferential shortening and twist mechanics,[42] whereas the subendocardial longitudinal mechanics are relatively spared (Table 4).

Table 3. Comparison of Myocardial Mechanics in HFPEF and HFREF

	HFPEF	HFREF
Longitudinal strain	Markedly decreased	Markedly decreased
Circumferential strain	Preserved/mild decrease	Markedly decreased
Radial strain	Preserved/mild decrease	Markedly decreased
Twist	Preserved	Markedly decreased
Untwist	Delayed/may decrease	Delayed and decreased
Global EF	Preserved	Markedly decreased

HFPEF, heart failure patients with preserved ejection fraction; and HFREF, heart failure patients with reduced ejection fraction.

Table 4. Effect of Physiological Variables on Left Ventricular Twist Mechanics in Comparison to Resting Healthy Young Controls

	Systolic Twist	Untwist Rate
HFNEF	↑	Normal or decreased
HFREF	↓	↓
Dilated cardiomyopathy	↓	↓
Transplanted hearts	↓	↓
Ventricular Noncompaction	Little or none	Little or none
Hypertrophic cardiomyopathy	↑	Normal or decreased
Constrictive pericarditis	↓	↓
Chronic myocardial ischemia	↓	↓
Aortic stenosis	↑	↓
Compensated mitral regurgitation	↑	↓
Decompensated mitral regurgitation	↓	↓
Congenital heart diseases and pulmonary hypertension	↓	↓

HCM indicates hypertrophic cardiomyopathy; HFNEF, hart failure with normal ejection fraction; and HFREF, heart failure with reduced ejection fraction.

Valvular Heart Disease

In patients with significant valvular disease, changes in twist mechanics depend on the degree of systolic dysfunction[43] (Table 4). In patients with aortic stenosis and preserved EF, subendocardial ischemia causes LVT to increase, contributing to the preservation of EF. However, untwist decreases and is associated with diastolic dysfunction and elevated LV end-diastolic filling pressures.[44] Aortic valve replacement either by surgery or by transcatheter (TAVI) in these patients normalizes the LVT,[43,45] although the development of significant aortic regurgitation post-TAVI abolishes this normalization. However, aortic stenosis patients with reduced EF have reduced LVT, signifying exhaustion of LVT compensatory mechanisms and LVT in these patients does not change post-TAVI.

In well-compensated mitral regurgitation, the increased preload enhances LVT.[46] Treatment of these patients with mitral valve repair surgery cause LVT values to return to normal.[47] Conversely, chronic mitral regurgitation in the presence of LV systolic dysfunction reduces LVT because of a decrease in the lever arm force of the epicardial fibers.[48] In the presence of LV systolic dysfunction, LV torsional parameters correlate with the degree of LV remodeling and the severity of MR[49].

Summary and Future Directions

The case presented in the clinical vignette illustrates the effects of the subendocradial dysfunction on LVT mechanics as the underlying mechanism of diastolic dysfunction. At rest, the patient showed an increased LVT, which seems to have compensated for the depressed global longitudinal strain, and thus the EF was relatively preserved. This might be explained by the development of subendocardial dysfunction related to longstanding untreated hypertension that increased the leverage of the subepicardial fibers. After exercise, there was a further reduction in global longitudinal strain, lack of increase in LVT, and reduction of early diastolic UTR and a worsening of the diastolic parameters. This also might be explained by the further worsening of the subendocardial dysfunction associated with the exercise induced sudden increase in blood pressure. Therefore, this case represents an example of the blunted exercise reserve mechanism in a hypertensive patient that leads to diastolic dysfunction and eventually heart failure with preserved ejection fraction.

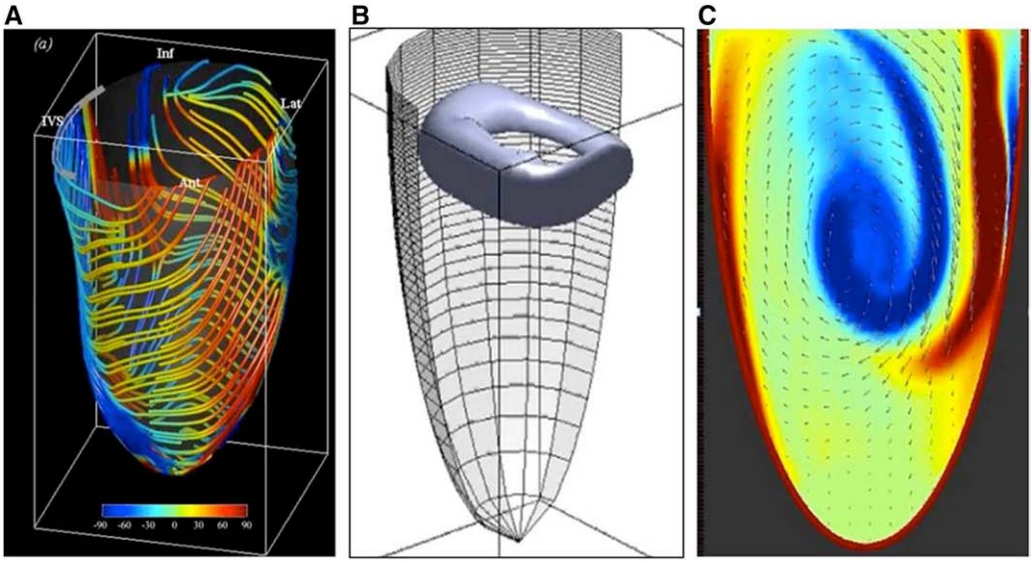

Figure 6. Integrated assessment of left ventricular (LV) deformation, twist, and LV fluid mechanics. **A**, Average diastolic fiber strain (derived from principal strain, which simultaneously integrates myocardial deformations in all directions, including LV twist[51]) reconstructed from 3-dimensional speckle-tracking endocardial surface strain. Strain lines are colored by their angles represented by the color bar on the bottom. **B**, Three-dimensional computer model showing the formation of doughnut-shaped vortex ring formed inside the LV cavity in early diastole because of LV untwisting. Although LV ejection is accompanied with wringing deformation, early diastole is characterized by formation of vortex rings. **C**, Two-dimensional cross section of the LV vortex showing the larger asymmetrical anterior rotation of the intracavitary vortex ring, which facilitates blood flow redirection toward LV outflow. Panels reconstructed, courtesy of Pedrizzetti et al.[51]

The case illustrated above thus provides evidence that the use of LVT measurements can provide mechanistic insights in the clinical assessment of myocardial diseases.

However, despite the growing evidence, the routine clinical use of these measurements has not yet been recommended.[50] This is related to the lack of standardization between various software and the challenges in obtaining basal and apical cross-sectional views. Both factors contribute to a great source of variability and necessitate standardization efforts to be undertaken by scientific consortiums. The European Society of Cardiovascular Imaging and the American Society of Echocardiography (ASE) have already initiated a strain standardization task force that endeavors to standardize the application of 2D-STE deformation imaging.

It is important to take into consideration that methods currently used to assess myocardial LVT mechanics may be challenged by image quality, ultrasound frequency, and frame rates. Importantly, 2D-STE measurements are not possible in a single image acquisition. 3D-STE is a possible solution to this problem, and also has potential application in the assessment of myocardial principal strain (Figure 6A), a newly developed concept of myocardial mechanics, which simultaneously integrates myocardial deformations in all directions, including torsional deformation, alleviating the need for multidirectional strain assessments.[51] However, 3D echocardiographic image acquisition is currently limited by low frame rates and reduced temporal resolution. This can be addressed with the use of the recently developed high-frequency echocardiographic systems, which represent a new platform for ultrasound-based assessment in humans with high temporal and spatial resolutions.[52] It has been recently suggested that blood motion inside the LV is characterized by diastolic vortex formation (Figure 6B and 6C) that accompanies the redirection of jet flow toward the LV outflow tract and has a crucial role in fluid dynamics and mechanical efficiency of LV contraction.[53] LV vortex formation is closely related to the sequence of LVT and untwist, thus, warranting future studies that focus on these relationships in health and disease.

Disclosures

None.

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KEY WORDS: diastolic heart failure ■ echocardiography ■ hypertension ■ left ventricular function ■ mechanics ■ torsion

Left Ventricular Twist and Torsion: Research Observations and Clinical Applications

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Correction

In the article by Omar et al, "Left Ventricular Twist and Torsion Research Observations and Clinical Applications," which appeared in the June 2015 issue of the journal (*Circ Cardiovasc Imaging.* 2015;8:e003029.), a correction was needed.

The authors discovered an error in Figure 2A and Figure 3C of the article; all arrows should be reversed.

The authors regret this error.

This correction has been made to the article, which is available at http://circimaging.ahajournals.org/content/8/6/e003029.

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