Diastolic dysfunction contributes significantly to the clinical syndrome of congestive heart failure in the settings of both preserved and impaired left ventricular (LV) systolic function. Over the past 20 years, the clinical diagnosis of diastolic dysfunction has been based largely on echocardiographic Doppler parameters of LV filling. The most important limitation of these parameters is their dependence on loading conditions. New practical and comprehensive methods of evaluating diastolic function are critically needed not only for diagnosis but also for testing emerging new therapies.

Recent work has elucidated both intracellular and extracellular mechanisms that contribute to diastolic dysfunction, and some progress has been made in finding new treatments for diastolic heart failure. Additional research has evaluated the contribution of LV architecture and its role in maintaining the normal contraction sequence that optimizes mechanical efficiency. The importance of systolic twist and the subsequent recoil that leads to diastolic untwisting was examined in the article by Notomi et al using tissue Doppler techniques in both normal subjects and patients with hypertrophic cardiomyopathy, at rest and during exercise.

The Left Ventricle Viewed as a Double Helix That Twists and Untwists

Leonardo DaVinci in the 16th century and Rushmore in the 1950s described the rotational motion of the left ventricle. In 1970, McDonald reported a study in which he placed radiopaque markers on the epicardium of patients undergoing closed mitral valvuloplasty. He demonstrated that the pre-ejection phase was characterized by descent of the base and a counterclockwise rotation of the epicardial surface of the left ventricle with a thrust of the apex toward the chest wall. In late systole, there was a slight clockwise rotation and apical retraction.

Using blunt dissection, Torrent-Guasp et al demonstrated that the ventricles consist of a single myofiber band starting at the right ventricle just below the pulmonary valve and forming a double helix extending to the left ventricle, where it attaches to the aorta (www.helicalheart.com). This architecture promotes systolic torsion. When viewed from the LV apex, the systolic rotation of the base is clockwise, and the rotation of the apex is counterclockwise. Torsion is defined as the difference between the basal and apical rotation. In diastole, the ventricle “untwists,” rapidly recoiling and creating diastolic suction. This “suction” increases the diastolic intraventricular gradient and the left atrial to LV gradient to promote early filling.

If the normal LV architecture has a major role in normal diastolic function, it stands to reason that the ventricle, remodeled in response to an injury such as myocardial infarction, would display diastolic dysfunction, in addition to systolic dysfunction.

Diastolic Function During Exercise

During exercise, the left ventricle increases its total output not only by increasing heart rate but also by augmenting LV stroke volume. LV stroke volume increases through an increase in both the end-diastolic volume and ejection fraction. However, the shorter diastolic filling period associated with the increased heart rate provides less time to accomplish increased filling. Thus, to increase end-diastolic volume without elevating diastolic pressure, there must be lusitropic enhancement and an increase in the early filling rate. Early diastolic elastic recoil is enhanced as a result of the increased force of systolic contraction with greater shortening. It also has been shown that β-adrenergic stimulation during exercise results in an increased rate of calcium uptake by the sarcoplasmic reticulum and accelerated myocardial relaxation. By these mechanisms, the early LV diastolic pressure is lower during exercise than at rest, and the pressure-volume curve shifts downward during exercise.

Using a method based on 2D imaging, Tischler and Niggl previously demonstrated that systolic twist is enhanced by 86% during exercise from ~10° to 18°. However, there are no data on the effect of disease processes that impair systolic twist during exercise and how they affect the early diastolic untwisting or on those that primarily affect diastolic function. The importance of the untwisting of the ventricle in early diastole and its enhanced rate in exercise is demonstrated by the findings of the study by Notomi et al.

Measuring Twist and Untwist

MRI tissue tagging has been used to measure twist and torsion of the heart. The limited temporal resolution limits its use primarily to resting studies. Nevertheless, important information on diastolic untwisting has been gleaned from MRI. It has been demonstrated that during isovolumic relax-
ation. ≈40% of the accumulated systolic torsion is released. In animals, the velocity of LV untwisting (recoil rate) has been correlated with invasive measurements of τ, the time constant of relaxation, under a variety of loading and isotropic conditions. Dong et al showed that the recoil rate is a measure of LV relaxation that is independent of preload as reflected in left atrial pressure. Interestingly, this same group of investigators showed that the diastolic dysfunction associated with normal aging was not explained by diminished recoil rate. The precise timing of rapid recoil associated with ventricular untwisting and the response to exercise have not been elucidated by MRI studies, in part because of the slow frame rate of acquisition.

### Tissue Doppler Applications for Measuring Twist and Torsion

In this issue of *Circulation*, Notomi et al use the concepts of LV systolic torsion and subsequent diastolic untwisting to further explore the mechanisms of diastolic filling during exercise. Echocardiograms were performed at rest and during exercise on 20 volunteers with structurally normal hearts and no history of hypertension. Tissue Doppler velocities of the septal, lateral, anterior, and posterior walls of the left ventricle were obtained in the short-axis view at the basal, mid, and apical levels. The measured tangential velocities were converted to angular velocities to estimate LV rotational velocity. These calculations were performed in both systole and diastole, therefore measuring both twisting and untwisting velocities, respectively, at the apex and base of the heart. LV torsion values, in degrees, were obtained by calculating the difference between apical and basal rotational velocities. These authors previously validated these techniques and calculations in 20 normal volunteers by comparing the tissue Doppler measurements to tissue-tagged MRI studies. They showed close correlation between results from their Doppler tissue imaging–based technique and MRI-based torsion data on the same patient. In addition to rotational velocity and torsion data, base-to-apex intraventricular pressure gradients (IVPGs) were obtained with color M-mode data. Increased IVPGs are one explanation for the diastolic suction of blood flow across the mitral valve and have been shown to increase with exercise.

The results of the study by Notomi et al support previous work showing that systolic torsion results from clockwise basal rotation with more vigorous counterclockwise apical rotation and provide new information on the timing and magnitude of the diastolic untwisting. The rapid transition to untwisting begins just slightly before the end of systole as marked by aortic valve closure, followed in time by the peak IVPG and then peak early diastolic filling. The relative timing of long-axis lengthening and short-axis expansion also were examined; these events occur simultaneously with or slightly after the peak E velocity, suggesting that they are a consequence rather than a cause of diastolic filling. These data suggest that the initiation of ventricular untwisting is an early and key mechanism that promotes early diastolic relaxation and early diastolic filling, possibly more important than the recoil of systolic basal descent.

During exercise, the degree of systolic LV torsion increased primarily as a consequence of increased apical counterclockwise rotation velocity. Untwisting began earlier, and velocities were markedly increased during exercise. The magnitude of increase in twisting and untwisting velocities during exercise was significantly greater than the corresponding changes in LV length and radius. This finding suggests that the enhanced diastolic suction during exercise results in large part from the more vigorous untwisting motion of the ventricle, especially at the apex. Not surprisingly, the measured IVPG, a marker for the suction phenomenon during diastole, was correspondingly increased with exercise in the normal subjects.

To explore the pathophysiological implications of these findings, Notomi et al then applied these methods of measuring LV rotation, torsion, and IVPGs in patients with hypertrophic cardiomyopathy (HCM) both at rest and during exercise. Interestingly, at rest, peak systolic torsion values in HCM patients were higher than in normal subjects. However, during exercise, the increased twisting and untwisting velocities so clearly demonstrated in normal subjects were blunted in HCM patients. The IVPG in HCM subjects increased with exercise but to a much smaller degree than in normal subjects. Moreover, the timing of untwisting was delayed within the cardiac cycle in these patients. These data suggest that a prominent mechanism for exercise intolerance in HCM is an inability to enhance diastolic untwisting, resulting in less diastolic suction, impaired LV filling, and increased left atrial pressure.

### Implications

In summary, this study elucidates mechanisms underlying lusitropic enhancement during exercise. The authors provide compelling evidence that a transition from LV twisting to LV untwisting is the mechanical event that initiates LV relaxation. Untwisting is followed by an appropriate increase in IVPG as a marker of diastolic suction and then by peak diastolic filling. Twisting and untwisting velocities are greatly enhanced during exercise, as are IVPGs, in the normal left ventricle but not in the myopathic ventricles of HCM patients. The tissue Doppler techniques used in this study offer greater temporal resolution than MRI and are performed more easily during exercise.

The measurements of twist, torsion, and IVPG obtained in the study by Notomi et al offer a more complex and detailed quantification of diastolic function. However, there is no new insight as to whether the mechanism is energy consuming or is the result of the release of elastic energy without energy consumption. It would be intriguing to study these measures in other conditions associated with diastolic dysfunction such as hypertensive heart disease and infiltrative disorders of the myocardium. The important role of apical torsional enhancement may explain the diastolic dysfunction observed in patients with apical aneurysms. Recent efforts to affect ventricular mechanics with surgical procedures that alter ventricular geometry such as the Dor procedure are likely to affect twist and torsion during both systole and diastole. MRI data have shown that systolic torsion is not restored after partial left ventriculectomy in patients with dilated cardiomy-
opathy despite clinical improvement. The effects of this restorative surgical procedure on diastolic untwisting should be studied with the tissue Doppler methodology used in this report. Additionally, measurements of twisting and untwisting velocities and diastolic suction could be followed clinically as a response to pharmacological therapy as our armamentarium expands to include agents that are targeted primarily at diastolic function.

**Bringing Twist and Untwist to the Clinical Echocardiography Laboratory**

The observations of this study could lead to important tools for clinical echocardiographers to use in their routine clinical assessment of diastolic function once the load independence of the variables has been confirmed. Moreover, as with other functional parameters that have preceded these measures of diastolic untwisting or torsion, simplified methodology that requires minimal offline analysis will be required. The most useful measures for understanding diastolic function will need to be identified among the proposed variables. Further exploration of other methods such as those based on myocardial speckle tracking that, unlike Doppler-based methods, are independent of angle is needed. Three-dimensional methods are likely to be more robust in measuring systolic and diastolic torsion, as suggested by the authors of this study.

With the “Twist,” Chubby Checker changed dancing forever by separating the dancers, freeing them to express themselves.20 Looking beyond traditional measures of diastolic function, echocardiographers may now be able to express twist, untwist, and torsion.

**Disclosures**

None.

**References**

New Untwist on Diastole: What Goes Around Comes Back
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