

Clinical Application of Left Ventricular Twist: A Twist in the Right Direction?

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Understanding the fundamentals of cardiac function in a clinical setting rests entirely on the evaluation of the systolic and diastolic properties of the heart. In the bygone years, invasive catheterization was used, particularly in animals, to investigate the systolic function of the left ventricle (LV) via the gold standard parameter, elastance, as the strongest marker of the LV systolic performance in a pressure-volume relationship algorithm. Similarly, the tracings of the left atrial pressures and studies of the time constant of relaxation (τ) were used to investigate the diastolic properties of the heart. Under normal circumstances at rest and during physical activity, the stroke output of the LV is maintained by keeping the left atrial pressure within 10 mmHg.

Over the last couple of decades, however, there has been a paradigm shift in terms of understanding the systolic and diastolic properties of the heart noninvasively using echocardiographic post-processing with one-dimensional tissue Doppler imaging, two-dimensional (2D) based non-Doppler speckle tracking echocardiography (STE), or cardiovascular magnetic resonance imaging (CMR) feature tracking (1). As CMR is deemed a white elephant, echocardiography remains the most widely available bedside choice for the quantification of the myocardial motion during all the phases of the cardiac cycle, not only during the systole and diastole but also during the isovolumic periods of contraction and relaxation (2). While one-dimensional strain is fast losing its edge over 2D strain, 3D speckle tracking (STE) may circumvent some limitations associated with 2D STE. 3D speckle tracking may circumvent some limitations associated with 2D STE. The latter two are the most valuable tools in our armamentarium to scrutinize the LV function along multiple planes, noninvasively.

The most exciting application of STE is its ability to negotiate myocardial mechanics in health and disease. The heart is composed of a uniquely designed myo-architecture in a complex helix of right-handed subendocardial and left-handed subepicardial fibers sandwiching a mid-

circumferential layer. This unique distribution can be studied using echocardiography-based shear wave imaging (3). Not only can STE unravel the motion of the heart along all these planes of myofibril orientation, generating invaluable strain data both online and offline along multiple domains (i.e. longitudinal, circumferential, and radial), but also it confers an assessment of the LV rotational mechanics in routine clinical situations such as acute myocardial infarction (4) and dilated cardiomyopathy (DCM) (5). Although 2D STE has been studied extensively, 3D STE may also provide principal strain in such clinical situations as hypertension (6) and heart failure (7). It is interesting to note that the principal strain (maximum force of contraction), mostly investigated through magnetic resonance imaging, along with the LV twist maintains the ejection fraction even to a supra-normal level when the longitudinal and circumferential strains are compromised in certain situations, for instance in the LV hypertrophy secondary to hypertension (8).

The LV twist is the result of the net sum of the basal clockwise rotation and the apical counterclockwise rotation. The principal contribution of the twist does come from the epicardial fibers as it counteracts the opposing motion of the subendocardial fibers. At the basal state, the different layers of the myocardium have different strain. During systole, there is a so-called equalization of strain across the whole myocardial fiber orientation, resulting in a comprehensive and efficient delivery of the stroke volume (9). Question naturally arises as to what role the LV twist plays in this final endpoint of the stroke output delivery. It is assumed that the LV twist is a sensitive marker of the contractile function of the outer (subepicardial) wall but did not contribute much to the overall ventricular function (10). Twist is, nonetheless, an important component of the systolic reshaping of the LV, without which the heart would certainly underperform as a pump. It really does not matter how much of the 70-milliliter stroke volume comes from the LV twist; this rotational deformation has been validated against

CMR-based tagging (11) and by sonomicrometry (12) and can hence be used clinically to evaluate the systolic function as well as the LV suction in the very early stage of diastole. Therefore, the LV twist and untwist have been applied clinically. Govind et al. (4) demonstrated that in their subjects with ST-segment elevation myocardial infarction, the LV torsion was greatly diminished and there were subtle systolic and diastolic abnormalities in hypertension (6, 12). The value of the LV twist was also studied in non-selective ambulatory patients attending the cardiology outpatient services in India and the results showed that the subjects had abnormal LV rotational diastolic parameters, albeit with a preserved LV ejection fraction (13).

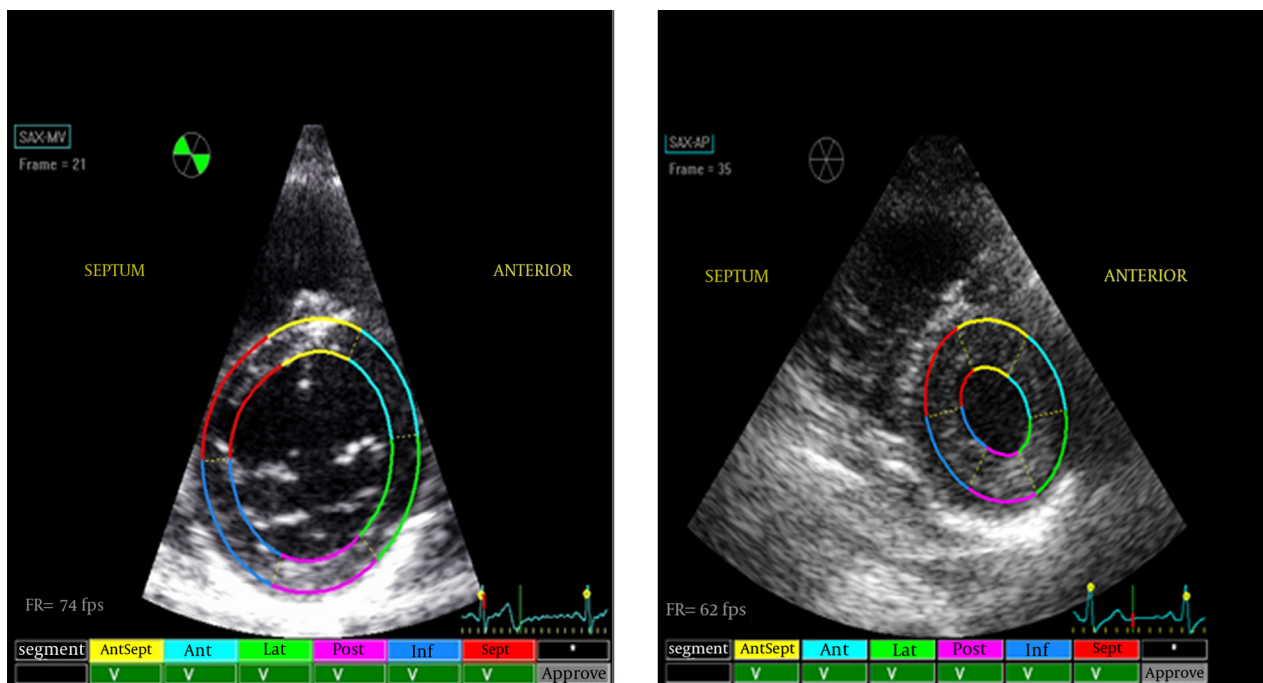
Left Ventricular Twist as Cardiac Adaptation?

Weiner et al. (14) designed a prospective, longitudinal study to determine whether endurance exercise training for 90 days would change the LV twist mechanics. The authors found a significant augmentation in the peak systolic LV twisting and untwisting rates after 90 days of endurance training and concluded that the systolic and diastolic LV rotational mechanics augmentation could be an “important and previously unrecognized component” of exercise-induced cardiac remodeling. Interestingly, however, the conventional LV systolic and diastolic variables remained unchanged. In contrast to this finding, Nottin et al. (15) showed that after an ultra-long duration

exercise in athletes, the LV torsional parameters were depressed with delayed diastolic suction compared with the pre-exercise data. However, it should be noted that in the latter study, the post-exercise torsion was measured immediately after exercise. The comparison between the pre- and post-exercise torsion was beautifully displaced in a twist-radial displacement loop.

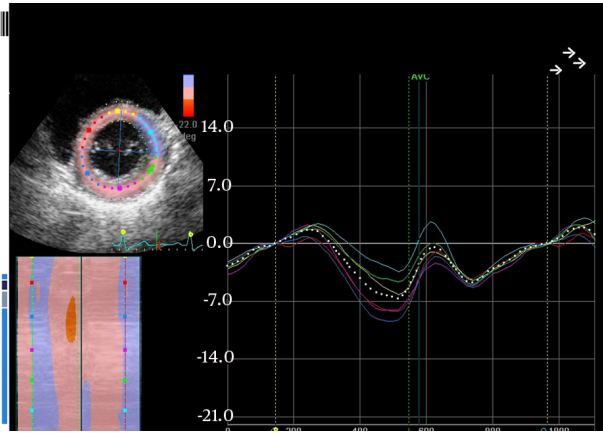
Another study (4) used a relatively new algorithm to compute twist, the morphology of which was slightly different from that of the more conventionally used algorithm. It is difficult to assess whether this new algorithm could be validated by hitherto non-existing, vendor-independent software to compute twist on a 2D plane, illustrating the simultaneous motion of the base and apex of the LV, as in Figure 1-4 showing 2D STE and Figure 5 showing an example of 3D STE-obtained LV twist. Industry and academia are seeking a unified nomenclature and a universally acceptable, guideline committee-approved algorithm. Be that as it may, this study is a welcome initiative to investigate the LV twist in DCM. One advantage of using DCM is that the allegedly falsely inflated ejection fraction value in the hypertrophied heart belies the compromised shortening value, particularly in pathological hypertrophic states. In DCM or in heart failure with a reduced LV ejection fraction, however, this mismatch is not seen and it, therefore, offers a better pathologic model to study twist. The authors deserve credit for that.

Figure 1. Manual Tracking of the Short-Axis Image at the Mitral Valve Level (Left) and Short-Axis Image at the Left Ventricular Apex (Right)



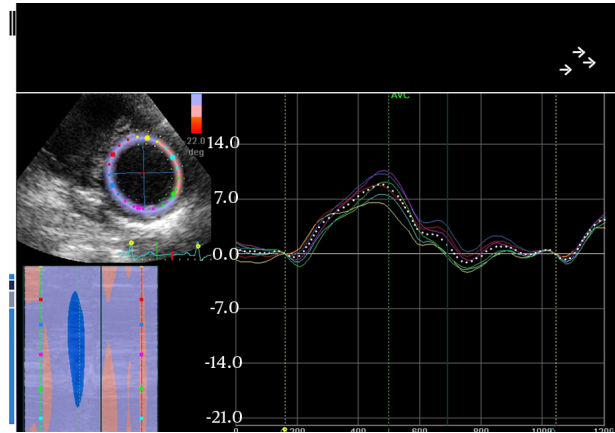
All the segments are valid (“V”).

Figure 2. Rotation at the Mitral level



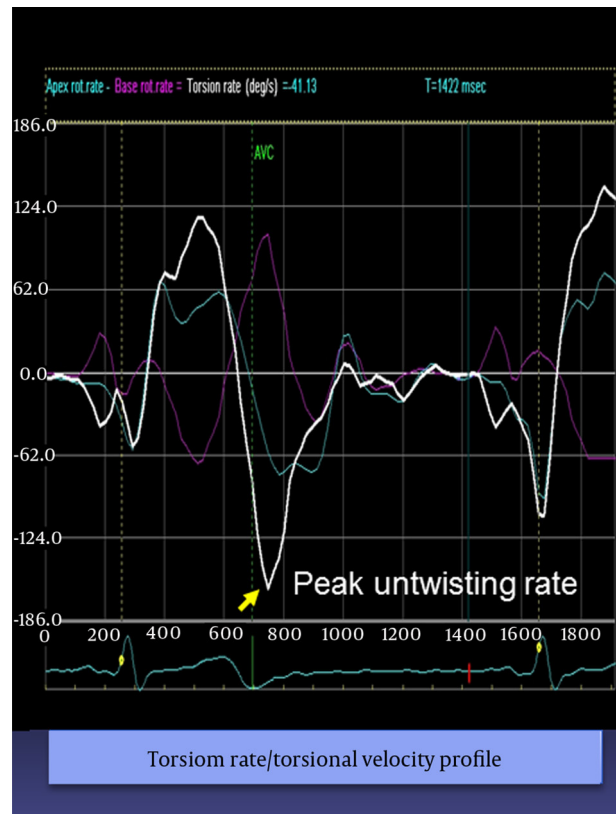
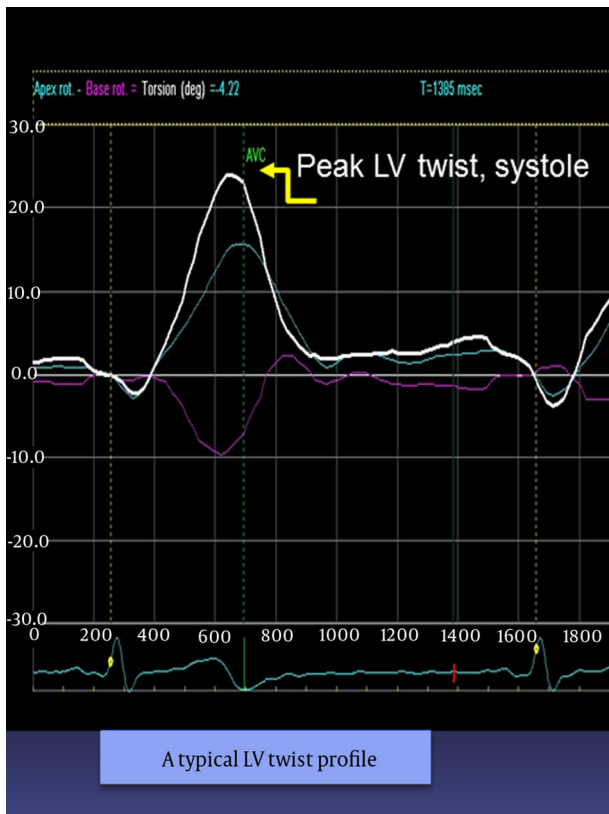
Clockwise motion of the LV base results in negative rotation profile.

Figure 3. Apical Rotation



Counterclockwise motion of the LV apex results in a positive rotation.

Figure 4. Left: LV Twist; Right: LV twisting and Untwisting rate (in this figure Torsion and Twist are considered synonymous).



By mathematical summation of the two components, it is possible to compute twist (degree) and its speed (degree/second) with reasonable tempo-spatial information.

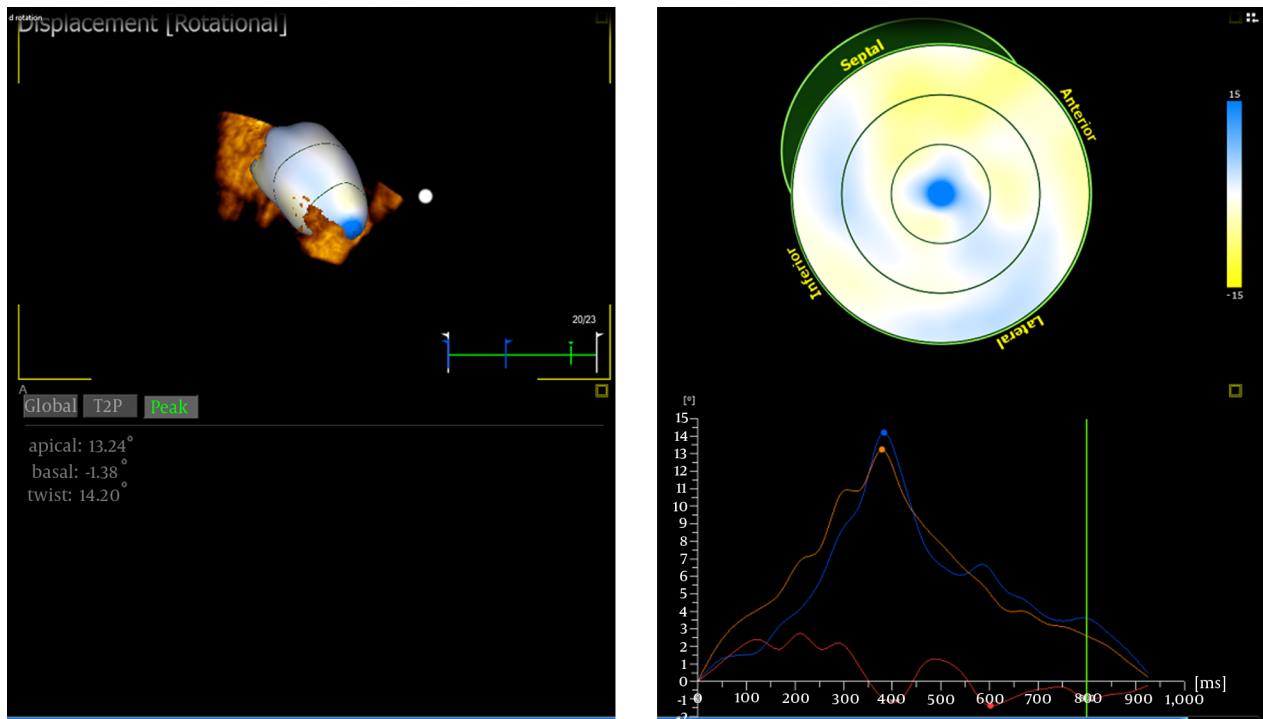


Figure 5. 3D STE-Obtained LV Twist

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