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Title: Left ventricular mechanics in advanced heart failure patients
Issue Date: 2015-11-03
CHAPTER 2

Role of left ventricular twist mechanics in the assessment of cardiac dyssynchrony in heart failure.

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Technologic innovations in cardiac imaging have provided new tools and algorithms for accurate assessment of left ventricular (LV) twist mechanics. This review provides a focused update on the incremental value of assessing LV twist mechanics in patients with heart failure (HF) and its potential role in characterizing response to cardiac resynchronization therapy (CRT). First, the findings are summarized from recent experimental and clinical studies that have specifically characterized the patterns of abnormal LV twist mechanics in HF. Next, the evolving application of LV twist is discussed in understanding response to CRT, elucidating the independent relationship between LV twist mechanics and reversal of LV remodeling at 6 months follow-up. Finally, the studies are addressed that underscore a critical relationship between LV lead position and changes in LV twist after CRT. These data suggests that the reversal of LV remodeling seen in HF patients following CRT primarily results from restoration of the global sequence of LV twist mechanics.
INTRODUCTION

Heart failure (HF) remains one of the major public health problems in developed countries. In United States, nearly 6 million patients have HF symptoms and 500,000 new patients are diagnosed yearly (1). Recently, important advances in HF therapy, such as cardiac resynchronization therapy (CRT), have improved the outcome of these patients (2). However, the prognosis still remains poor with a 5-year mortality of 42.3% after hospitalization for HF (1).

LV rotation, twist and torsion are important aspects of the cardiac mechanics. The term rotation is referred to the rotation of LV short-axis sections. Due to the spiral architecture of LV myofibers, the rotation of LV apex and base are counterclockwise and clockwise, respectively, as viewed from the LV apex. The opposite rotation of LV apex and base leads to a LV systolic wringing motion during systole referred to as twist or torsion. In particular, LV twist is the net difference at isochronal time points between apex and base in the rotation angle along LV longitudinal axis, whereas LV torsion is LV twist indexed to the distance between LV apex and LV base (3). This peculiar characteristic of the LV contributes significantly to LV systolic function, in addition to myocardial shortening and thickening.

Following a brief overview of physiology of LV rotational mechanics, an in-depth discussion is provided on different LV twist patterns in systolic HF and the evolving role of LV twist as a marker of LV-dyssynchrony for understanding response to CRT.

NORMAL LV TWIST MECHANICS

In the normal heart, the myofiber geometry of the LV changes gradually from a right-handed helix in the subendocardium to a left handed helix in the subepicardium. Taber et al. (4) explored the impact of this changing transmural myofiber orientation on LV rotational mechanics in a one-layer cylindrical model that consisted of obliquely aligned muscle fibers embedded in an isotropic matrix. The contraction of the epicardial fibers rotated the apical end of the model in the counterclockwise direction and the base in the clockwise direction. Conversely, shortening of the subendocardial fibers rotated the apex and base in clockwise and counterclockwise directions respectively. When both layers are coupled to contract simultaneously, a larger radius of rotation for the outer epicardial layer resulted in the epicardial fibers having a mechanical advantage in dominating the overall direction of rotation. The endocardial layer does provide some opposition to epicardial motion. This opposing action ensures that epicardial and endocardial sarcomere shortening in all directions are equilibrated during ejec-
tion, resulting in an optimal distribution of LV stress and strain (5). Elimination of twist decreases epicardial shortening at the expense of an increase in endocardial shortening. This in turn increases endocardial stress and strain, which increases oxygen demand and reduces the efficiency of LV systolic performance.

Taber’s model also provides explanation for the temporal changes in the sequence of LV twist during a cardiac cycle. The initial shortening of subendocardium causes a brief clockwise rotation of LV apex during the isovolumic contraction (6). Subsequent transmural spread of electrical activation, results in simultaneous shortening of subendocardial and subepicardial fibers. Due to the subepicardial fibers having a larger moment arm, the direction of rotation is shifted towards a counterclockwise rotation for the LV apex and a clockwise rotation for the LV base (Figure 1).

In the subepicardium, this twist supports contraction in the principal fiber direction. In the midwall, the twist enhances shortening in the circumferential direction.

Twist deformation of the LV wall causes fiber rearrangement that maximizes the LV wall thickening. In particular, twisting and shearing of the subendocardial fibers also deforms the matrix and results in storage of potential energy by compression of cardiac proteins such as titin (6). The potential energy stored in the titin is subsequently unleashed during diastole, aiding myocardial relaxation and diastolic filling.

**Figure 1. Mechanism of left ventricular twist**

Left ventricular (LV) fiber orientation changes from a right handed helix in the subendocardium to a left handed helix in the subepicardium (A). During isovolumic contraction (IVC), circumferential components of force (arrows) are generated by endocardial fiber shortening, which rotates the LV about the long axis clockwise as viewed from the apex (B). During ejection, shortening of subepicardial fibers wrapped in an opposite, left-handed helix, rotates the LV counterclockwise (C). Twisting force by epicardial shortening overcomes the forces of subendocardial shortening because the torque of the epicardial force is larger due to a greater radius of the epicardial fibers from the central LV long axis.
Factors Affecting LV Twist

Alterations in preload, afterload and contractility have been shown to alter cardiac rotation (1). Loading mechanics influences twist through changes in LV end-diastolic and end-systolic volumes. The directly proportional relationship between torsion and LV end-diastolic volume and the inversely proportional relationship between torsion and end-systolic volume illustrate the volume dependency of LV torsion. Like changes in loading conditions, increasing contractility increases LV twist; for example, positive inotropic interventions such as dobutamine infusion and paired pacing, greatly increase LV twist, whereas negative inotropic interventions markedly reduce twist (1).

Moreover, the LV twist increases gradually from infancy to adulthood. Notomi et al. (7) assessed LV torsion and twisting velocities in individuals from 9 months to 49 years and found that with advancing age there was an increase in LV torsion and untwisting velocity. Several other investigations examining older individuals have shown LV torsion to be maintained or increased compared with younger adults. It has been proposed that endocardial function is more likely to reduce with age due to the subendocardium’s greater susceptibility to fibrosis and/or subclinical reductions in perfusion. As per Taber’s model, the reduced endocardial function would result in less opposition to the dominant epicardial action causing increase in rotation. The finding of reduced subendocardial function and increased torsion in older individuals results in preservation of global LVEF, suggesting a compensatory mechanism that helps to preserve global LVEF despite the presence of subendocardial dysfunction.

LV TWIST IN THE DYSSYNCHRONOUS, FAILING VENTRICLE

LV twist is emerging as an important parameter of LV systolic function. Several authors previously reported a significant correlation between LV twist and LVEF, the most commonly used index of LV systolic function in clinical practice (1). However, there is increasing evidence that LV twist is superior to LVEF in characterizing hemodynamic aberrations in patients with HF. For example, Kim et al. (9), in a recent experimental study, reported a strong correlation between dP/dt$_{max}$ (an invasive, relatively load-independent, measure of LV contractility) and LV twist ($R^2 = 0.747$, $p <0.001$); however, the correlation between dP/dt$_{max}$ and LVEF, despite significant, was weaker ($R^2 = 0.408$, $p <0.001$). This observation is related to specific differences in LV twist and LVEF: LV twist is an index of systolic myocardial deformation, while LVEF simply reflects LV volume reduction during systole.
In particular, the LV torsional deformation, related to the spiral architecture of LV myofibers, permits the generation of LVEF ≥60% from myofibers that can shorten by only 15%; otherwise, simple longitudinal or circumferential shortening would not allow LVEF higher than 30% (10, 11). Besides being a sensitive indicator of myocardial performance, the LV rotational mechanics appear strongly related to the sequence of LV depolarization as well; the propagation of the electrical cardiac activity is indeed significantly related to the spiral architecture and the anisotropic properties of cardiac myofibers (11). The assessment of LV twist, therefore, may provide more in-depth understanding of the pathophysiology of HF, as compared to the traditional parameters of LV systolic function.

The Ischemic versus the Non-ischemic Failing Ventricle

Significant alterations of LV rotational mechanics have been observed in patients with previous myocardial infarction (MI) and chronic ischemic and non-ischemic HF.

Myocardial infarction. Several studies showed an impairment of LV twist after MI (5, 8). The observed reduction of LV twist correlates with the reduction of LVEF (being more pronounced when LVEF is <45%) and the number of dysfunctional myocardial segments. In addition, Gjesdal et al. (13) recently observed a significant correlation between LV twist and the infarct mass \( (r = -0.59, \ p < 0.001) \). The injury caused by the infarction to the LV myofiber architecture, may explain these findings. Indeed, Wu et al. (14), using diffusion tensor magnetic resonance imaging, observed an increase of left-handed myofibers and a decrease of right-handed myofibers in the infarct area; the extent of these changes was associated to the infarct size. Interestingly, opposite changes were observed in the remote zone, likely representing an adaptive response to increased wall stress.

Ischemic versus non-ischemic HF. As compared to MI patients, HF patients present an even more pronounced impairment of LV rotational mechanics, irrespective of HF etiology as result of reduction of both LV basal and apical rotation (Figure 2) (8, 15-17). In particular, the typical counterclockwise rotation of the LV apex may be completely abolished, or even reversed in a clockwise rotation. Recently, in a population of advanced HF patients with prolonged QRS duration, Bertini et al. (18) showed a modest but significant correlation between LV twist and LVEF \( (r = 0.53, \ p < 0.001) \). This finding supports the hypothesis that LVEF and LV twist are not identical parameters, and LV twist may provide incremental information on LV systolic performance.

According to previous experimental studies, several mechanisms may explain the impairment of LV twist in HF patients. First, as demonstrated by Taber et al. (4), LV
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Dilatation and thinning, present in dilated cardiomyopathy, equalize the radii of the subepicardial and subendocardial layers; as a result, the mechanical advantage of the subepicardial myofibers (the major determinants of LV twist under physiologic conditions) is reduced. Consequently, LV twist decreases with increasing cavity volume. Second, the long-lasting processes determining dilated cardiomyopathy and eccentric hypertrophy, cause myofibers disarray and alterations in myofibers angle \((^{16}\)) . These phenomena eventually lead to the loss of the physiological spiral architecture of the LV and to the impairment of LV twist \((^{19}\)) . Last but not least, slowed transmural fiber activation, related to fibrosis and remodeling of gap junctions, may delay the activation of the epicardial myofibers, determining an initial clockwise twist (because of the unopposed rotation of the endocardial myofibers) and finally an impaired peak LV twist \((^{20, 21}\)) .

According to these observations, it has been postulated that surgical techniques able to restore a more physiological shape of the LV would improve the LV torsional deformation \((^{17}\)) . Indeed, in a preliminary study of 26 patients with ischemic dilated cardiomyopathy, LV reconstruction surgery improved LV twist in the patients with more severely impaired LV twist at baseline \((^{17}\)) ; these patients showed also significantly greater improvement of LVEF after surgery as compared to the patients with

Figure 2. Left ventricular twist in acute myocardial infarction and ischemic versus non-ischemic heart failure.
Examples of left ventricular (LV) twist assessed with speckle tracking echocardiography in acute myocardial infarction (MI), and chronic ischemic versus non ischemic heart failure (HF). Of note, LV twist is markedly reduced in HF patients as compared to acute MI patient.
relatively more preserved LV twist at baseline (ΔLVEF 15 ± 8% vs. 6 ± 8%, p = 0.005) (17).

**Relation LV Twist-LV Dyssynchrony**

An effective LV pumping function requires the combination of preserved LV architecture and a preserved electrical conduction system. The presence of an abnormal activation sequence of the ventricles (e.g., right ventricular apical pacing, right or left bundle branch block) results in a slower spread of the electrical breakthrough across the myocardium and in a dysynchronous mechanical activation of the ventricles (22). In addition, the anisotropy of the LV myocardium determines the propagation of the electrical wavefront. As previously described, (12, 23) activation of the LV includes the development of a potential over the lateral-apical region which reflects endocardial-to-epicardial propagation of the LV free-wall activation front. Subsequently this epicardial potential is seen to migrate from the lateral LV apex toward the posterolateral base. The propagation is faster in the longitudinal direction of the myofibers rather than across in the circumferential cross-fiber direction due to the higher density of gap junctions concentrated in the intercalated disks along the longitudinal axis, as compared to the cross-fiber densities (12). In the remodeled, failing LV this particular architectural pattern may be distorted, with loss of anisotropy and gap junctions, resulting in a slower conduction of the electrical excitation.

Several experimental studies have demonstrated the deleterious effects of asynchronous ventricular activation on LV performance and the relation between the LV activation pattern and LV twist (24-28). Prinzen et al. (26) showed that ectopic activation induced asynchronous electrical activation and, subsequently, asynchronous cardiac motion (mechanical asynchrony). Interestingly, mechanical asynchrony was larger than electrical asynchrony because the time interval between the electrical activation and the onset of fiber shortening was more prolonged at the most delayed mechanical activated segments. Afterwards, changes in myofibers work within the LV wall were evaluated during right ventricular and LV pacing in normal hearts of dogs. Thus, both pacing modes determined a pronounced redistribution of midwall fiber shortening and work, with 50% decrease in myofiber work at the paced regions (hypofunctioning regions) and 150% increase at the remote areas (hyperfunctioning regions). These regional changes resulted in significant reductions in LV pump function, particularly when pacing from the right ventricle (27). Recently, Delgado et al. (29) compared the effects of right ventricular apical pacing on LV twist in 25 patients without structural heart disease. With the use of 2-dimensional speckle tracking imaging, the authors demonstrated that right ventricular apical pacing induced a
dyssynchronous mechanical activation of the LV, as measured by radial strain (from 21 to 91 ms, p<0.001) and a subsequent significant decrease in LV global longitudinal shortening (from -18.3 ± 3.5% to -11.8 ± 3.6%, p<0.001) and LV twist (from 12.4 ± 3.7° to 9.7 ± 2.6°, p=0.001; Figure 3).

Finally, two recent studies pointed out the relationship between LV-dyssynchrony and LV twist in advanced HF patients with prolonged QRS duration (18, 30). A first study showed that the extent of LV-dyssynchrony was inversely related to LV twist (18). Subsequently, these results were extended in another study demonstrating that LVEF and LV-dyssynchrony were both independently correlated to LV twist (30). This observation further underscores that LV twist is not only a parameter of LV function, but also reflects the extent of LV (dys)synchrony.
LV TWIST IN CRT

As previously indicated, LV mechanics and particularly LV twist are strictly dependent on electro-mechanical activation and are influenced by different pacing modalities \((24, 25, 31)\). However, thus far, data on the effects of CRT on LV twist are limited \((18, 30, 32)\).

Particularly, abnormal rotational mechanics in advanced HF patients with prolonged QRS duration may result from 2 different conditions that can also co-exist: 1) absolute reduction of LV apical and basal rotation (and consequently of LV twist), due to an impaired myocardial contractility; 2) dyssynchronous contraction of LV apical and basal regions, due to an altered pattern of LV electro-mechanical activation.

Figure 4. Left ventricular twist in the synchronous and dyssynchronous failing left ventricle.

Example of left ventricular (LV) twist in two patients with dilated cardiomyopathy and severe LV dysfunction (LV ejection fraction <30%).

Example of patients with synchronous (Panel A) and with dyssynchronous LV contraction LV (Panel B). In both the synchronous (Panel A) and the dyssynchronous LV (Panel B), the curves of the LV rotational parameters reveal reduced LV twist. Of note, the peaks of apical and basal rotation occur almost at the same time interval in the synchronous LV (Panel A), whereas they occur at different time intervals in the dyssynchronous LV (Panel B). In particular, in the dyssynchronous LV (Panel B) apical rotation is markedly earlier as compared to the basal rotation, which may result in further worsening of LV twist.
activation (Figure 4). Consequently, CRT, leading to a more physiologic electrical depolarization and mechanical contraction of the myofibers, has the potential to improve rotational mechanics in these patients.

Global Changes in LV twist after CRT

All the available studies are based on 2-dimensional speckle tracking echocardiography that, unlike tagged magnetic resonance imaging, allows the analysis of rotational parameters also after device implantation.

Recently, Zhang et al. (32) studied 39 patients scheduled for CRT, measuring LV twist at baseline and 3 months after implantation. At baseline, peak LV twist was significantly reduced in the HF patients as compared to normal controls (6.8 ± 4.2° vs. 16.2 ± 5.5°, p < 0.001). The authors also noted that in some patients, the presence of apical and/or basal segments showed a paradoxical rotation (clockwise for the apex and counterclockwise for the base), indicating a more compromised rotational mechanics. However, at short-term follow-up the authors could not detect any improvement of LV twist after CRT, although a significant increase of LVEF was observed (from 28.1 ± 6.7% to 35.0 ± 9.4%, p < 0.001).

Different findings were reported by Sade et al. (30) that studied the acute effect of CRT on 33 patients. At baseline, LV twist was significantly reduced as compared to normal controls either for ischemic and non-ischemic HF patients and correlated well with LVEF and radial dyssynchrony. A significant improvement of LV rotational

![Figure 5. Progressive improvement of LV twist induced by CRT.](image-url)

A significant and progressive improvement of LV twist was observed immediately after CRT and at 6 months follow-up.
mechanics was observed immediately after CRT. These controversial results may be related to the potential role of the LV lead position in determining LV twist pattern. However, no data about LV lead position were reported in these studies.

A more recent study (18) reported the acute and long-term effects of CRT on LV twist exploring also the influence of LV lead position. Specifically, in a group of 80 HF patients candidates to CRT a significant and progressive improvement of LV twist was observed immediately after implantation and at 6 months follow-up (Figure 5).

Responders versus Non-responders

The effect of CRT on rotational mechanics is more evident if the evaluation is performed according to the presence of LV reverse remodeling. Sade et al. (30) evaluated the changes in LV twist in 33 HF patients treated with CRT. Responder patients (with a reduction in LV end-systolic volume >10%) had an improved LV twist (from $1.5 \pm 2.8^\circ$ to $6.3 \pm 3.6^\circ$, p<0.0001). Conversely, in non-responders LV twist did not change or tended to worsen (from $5.3 \pm 3.1^\circ$ to $2.0 \pm 3.4^\circ$). Similarly in a more recent study (18), a significant improvement in LV rotational mechanics was noted only in patients who showed LV reverse remodeling (responders), both at the acute and long-term follow-up. In particular, peak LV twist progressively improved in responders during follow-up, whereas in non-responders a gradual deterioration of peak LV twist was observed (Figure 6). Furthermore, at the multivariable logistic regression analysis, in which LV-dysynchrony and function parameters were included, absolute difference in LV twist immediately after CRT was the strongest predictor of response to CRT at 6 months follow-up (OR = 1.837, 95%CI = 1.378-2.449, p <0.001).

These findings suggest that CRT may (partially) restore LV twist, possibly by providing a more physiologic electrical depolarization and mechanical contraction of the myofibers.

LV twist and LV lead position

LV lead position is considered a potential tool to increase CRT response rate. In clinical scenarios, the optimal site for LV pacing in patients receiving CRT remains controversial. Previous studies indicated that patients with a (postero-)lateral LV lead position and patients with a LV lead located close to the region with the latest mechanical activation do not only derive more benefit in restoring systolic LV function, but also tend to have superior long-term survival after CRT (33-36). The different patterns of LV depolarization induced by different LV lead positions may markedly change
Figure 6. Left ventricular twist changing in CRT responders and non-responders.

Panel A: Example of responder to cardiac resynchronization therapy (CRT). Peak left ventricular (LV) twist increases progressively from baseline to 6-month follow-up. In this example, the improvement of LV twist is mainly due to the improvement of LV apical rotation over time. Immediately after CRT, LV twist increases secondary to an improved electro-mechanical activation of the LV. Further improvement is observed at 6-month follow-up when LV reverse remodeling has also occurred. The lower panel shows the improvement in left ventricular ejection fraction (LVEF) and reduction in left ventricular end-systolic volume (LVESV) after 6-month follow-up.

Panel B: Example of non-responder to CRT. Peak LV twist declines progressively from baseline to 6 months follow-up. In this example, the main determinant of the reduction in LV twist is the deterioration of LV apical rotation. Indeed, the direction of LV apical rotation is reversed (negative red dashed curve) immediately after CRT and at 6 months follow-up. Here, the apical and basal levels have the same direction of rotation which results in a worsening of LV twist. At 6 months follow-up a reduction in LV basal rotation is also observed, which contributes to a further deterioration of LV twist. The lower panel shows the parallel worsening in LVEF and LVESV after 6 months follow-up.
the cardiac mechanics (12). Thus, in CRT patients, the magnitude of LV twist may be related to the LV pacing site. However, there is currently minimal data addressing this issue. Firstly, experimental studies showed that LV twist was influenced by the pacing mode (atrial, right and biventricular pacing) (14, 25, 31). For example, Sorger et

![Figure 7. Left ventricular twist versus left ventricular lead position.](image)

**Panel A.** Example of responder to cardiac resynchronization therapy (CRT) with the left ventricular (LV) lead placed in a (postero-)lateral vein with an apical position. Biplane fluoroscopy (left) displays the LV lead position. Particularly, the left anterior oblique (LAO) view shows the LV lead in the (postero-)lateral vein whereas the postero-anterior (PA) view shows the LV lead in an apical position. Peak LV twist increased from 3.8° at baseline to 10.6° at 6-month follow-up. LV ejection fraction (LVEF) improved from 24% at baseline to 38% at 6-month follow-up. In this patient, pacing close to the LV apical region may produce a more physiological pattern of electro-mechanical activation, resulting in a significant improvement in LV twist.

**Panel B.** Example of non-responder with the LV lead placed in a lateral vein (LAO view) with a basal position (PA view). Peak LV twist decreased from 9.4° at baseline to 4.7° at 6-month follow-up. LVEF decreased from 30% at baseline to 26% at 6-month follow-up. In this patient, pacing close to the LV basal region may induce a further worsening of the electro-mechanical activation with a significant worsening of LV twist.
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al. (25) evaluated the changes in LV twist during pacing from three different locations: right atrium, right ventricular apex and base of the LV free wall. Biventricular pacing with LV lead placed at the basal level of lateral wall, similarly to apical right ventricular pacing, worsened LV twist as compared to a more physiological electrical stimulation (i.e. right atrial pacing).

A recent study (18) explored the change in LV twist after CRT in relation to different LV lead positions in the (postero-)lateral veins. Interestingly, the authors observed that patients with LV leads positioned in mid-ventricular and apical regions exhibited a larger increase in systolic function with a significant increase in LV twist as compared to patients with LV leads positioned in the basal regions of the LV free wall (Figure 7). Possibly, LV pacing sites that yield the largest improvement in LV twist may likely determine a more efficient cardiac contraction with subsequent improvement of LV energetic (37). Similar results were obtained in an experimental study in a canine HF model, reporting that the mid-apical part of the LV free wall was the optimal stimulation site (38). These findings could be explained by the direction of cardiac depolarization, traveling from the apex towards the base in the normal heart (12, 39). Therefore, pacing close to the LV apex may replicate a more physiological pattern of LV depolarization and subsequent mechanical activation, leading to a significant improvement in LV twist (18, 25). Furthermore, as the myocardial wall is thinner in the LV apex compared to LV base (40, 41), pacing leads positioned near the apex are closer to the Purkinje network. This results in a faster electrical propagation of the cardiac pulse and subsequently a more synchronous LV contraction.

These are early data derived from small experimental and clinical studies, therefore larger multicenter studies are needed to confirm these findings.

FUTURE DIRECTIONS

Thus far, several indices of mechanical dyssynchrony have been proposed to select candidates for CRT. The analysis of LV twist may provide a more comprehensive evaluation of LV mechanics and may help to understand the effects of CRT in HF patients. Moreover, at present, CRT response relies on changes in clinical status, LV reverse remodeling and improvement in LVEF. In this regard, LV twist analysis may be incremental to changes in LV volumes and LVEF to characterize and define CRT response. Recently, Sade et al. (30) proposed to quantify the magnitude of LV twist at aortic valve closure timing as good index to predict CRT response, superior to LV-dyssynchrony. Furthermore, pioneer studies showed that an improvement of LV twist early after CRT predicts a reduction of LV volumes after 6 months (18). Future studies are warranted to
elucidate whether the magnitude and/or the specific pattern of baseline LV twist and immediate changes in LV twist after CRT may be used as a more sensitive index for the identification of CRT responders.

Currently, 2-dimensional speckle tracking echocardiography permits reliable assessment of LV twist mechanics (42). Furthermore, different authors reported a good reproducibility of the assessment of LV twist with 2-dimensional speckle tracking (18, 43, 44). However, 2-dimensional speckle tracking echocardiography has some limitations for the assessment of LV twist mainly related to the acquisition of LV apical short-axis images. This may be technically difficult and is highly dependent on the acoustic window and the through-plane motion, particularly at the basal level, that may affect accuracy of the measurement of LV rotational parameters. Recently developed 3-dimensional speckle tracking analysis may partially overcome these limitations and may provide even more global characterization of LV twist mechanics (45). Future technical advances will lead to improved accuracy and easier implementation of this technique in the clinical setting.

At present cardiac magnetic resonance remains the referral technique for the assessment of LV twist mechanics although its use is limited by availability and the presence of devices (pacemakers, internal cardioverter-defibrillators).

**SUMMARY AND CONCLUSION**

LV twist mechanics is a promising tool for characterizing the pathophysiology of HF. In advanced systolic HF, the rotational parameters are severely deteriorated and may be improved by restoring electro-mechanical activation through CRT. An immediate improvement in LV twist after CRT may be a good surrogate of a more physiological LV depolarization, and is independently related to reversal of remodeling after CRT. Finally, LV lead position is important for modifying the extent of LV twist after CRT; in particular pacing sites which provide the greatest improvement of LV twist likely determine the largest reversal of LV remodeling after CRT.
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