

Role of left ventricular twist mechanics in cardiomyopathies, dance of the helices

Floris Kauer, Marcel Leonard Geleijnse, Bastiaan Martijn van Dalen

Floris Kauer, Marcel Leonard Geleijnse, Bastiaan Martijn van Dalen, Erasmus University Medical Center Rotterdam, The Thoraxcenter, 's Gravendijkwal 230, 3015 CE Rotterdam, The Netherlands

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Correspondence to: Bastiaan Martijn van Dalen, MD, PhD, Erasmus University Medical Center Rotterdam, The Thoraxcenter, 's Gravendijkwal 230, Room BA 302, 3015 CE Rotterdam, The Netherlands. b.m.vandalen@erasmusmc.nl
Telephone: +31-10-4633533
Fax: +31-10-4635498

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Abstract

Left ventricular twist is an essential part of left ventricular

function. Nevertheless, knowledge is limited in "the cardiology community" as it comes to twist mechanics. Fortunately the development of speckle tracking echocardiography, allowing accurate, reproducible and rapid bedside assessment of left ventricular twist, has boosted the interest in this important mechanical aspect of left ventricular deformation. Although the fundamental physiological role of left ventricular twist is undisputable, the clinical relevance of assessment of left ventricular twist in cardiomyopathies still needs to be established. The fact remains; analysis of left ventricular twist mechanics has already provided substantial pathophysiological understanding on a comprehensive variety of cardiomyopathies. It has become clear that increased left ventricular twist in for example hypertrophic cardiomyopathy may be an early sign of subendocardial (microvascular) dysfunction. Furthermore, decreased left ventricular twist may be caused by left ventricular dilatation or an extensive myocardial scar. Finally, the detection of left ventricular rigid body rotation in noncompaction cardiomyopathy may provide an indispensable method to objectively confirm this difficult diagnosis. All this endorses the value of left ventricular twist in the field of cardiomyopathies and may further encourage the implementation of left ventricular twist parameters in the "diagnostic toolbox" for cardiomyopathies.

Key words: Left ventricular mechanics; Left ventricular twist; Cardiomyopathy

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Core tip: Left ventricular twist is an essential part of left ventricular function. Nevertheless, knowledge is limited in "the cardiology community" as it comes to twist mechanics. It has become clear that increased left ventricular twist in for example hypertrophic cardiomyopathy may be an early sign of subendocardial (microvascular) dysfunction. Furthermore, decreased left ventricular twist may be caused by left ventricular

dilatation or an extensive myocardial scar. Finally, the detection of left ventricular rigid body rotation in noncompaction cardiomyopathy may provide an indispensable method to objectively confirm this difficult diagnosis. All this endorses the value of left ventricular twist in the field of cardiomyopathies and may further encourage the implementation of left ventricular twist parameters in the “diagnostic toolbox” for cardiomyopathies.

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INTRODUCTION

As early as the 16th century Leonardo da Vinci^[1,2] wrote about the twisting deformation of the heart and Richard Lower compared the myocardial contraction with “the wringing of a linen cloth to squeeze out the water” in his observations of myocardial contraction in 1669^[3,4]. A complex spiral architecture is the mechanical basis for this wringing motion^[5,6]. The left ventricle comprises of obliquely orientated multiple layers of cardiomyocytes, transforming from a subendocardially located (smaller-radius) right-handed helix to a subepicardial (larger-radius) left-handed helix.

This helix generates a torsional motion pattern caused by rotation in a clockwise direction (as seen from the apex) at the level of the mitral valve (basal level) and counter clockwise rotation of the apex (apical level). This twisting deformation performs a fundamental part in the mechanical efficiency of the heart resulting in a 60% ejection fraction with only 15% fibre shortening^[7]. Furthermore, left ventricular untwisting is essential in actively aiding diastolic filling^[8]. The physiology of left ventricular twist and changes of left ventricular twist in different cardiomyopathies are reviewed in this paper.

ASSESSMENT OF LEFT VENTRICULAR TWIST

By his first description of left ventricular twist, Leonardo da Vinci^[1,2] has been a constant inspiration for scientists in their pursuit to comprehend the functioning of the human heart. Nevertheless, reliable quantitative measurement of left ventricular twist in a non-invasive manner has not been possible until recently.

Speckle tracking echocardiography is based on automated tracking of a specific portion of myocardial tissue being visualized by a pattern of gray values, a speckle pattern, on an ultrasound image. These gray values are the result of the analysis of the reflection of ultrasound interfering with the myocardial tissue.

Therefore, movement of speckle patterns represent motion of myocardial tissue^[9].

In case of a suitably high frame rate, the pattern of speckles is conserved from frame to frame^[10]. By following a specific pattern of speckles, the motion of the corresponding myocardial segment can be tracked, thus allowing to quantify deformation of the myocardium and, as a function of time, deformation rate (Figure 1). Several validation studies^[11,12] showed good correlation between left ventricular twist assessment by commercially available speckle tracking software and magnetic resonance imaging. Also, speckle tracking derived left ventricular twist has been shown to be feasible and reproducible, and may thereby be used as a method to follow-up patients^[13].

PHYSIOLOGY OF LEFT VENTRICULAR TWIST

After the first description of left ventricular twist by Da Vinci, it lasted until the late 1960s before a more detailed description of twist was provided by Streeter *et al*^[5] in a study of post-mortem canine hearts. Generally, myofibre position changes gradually from +60 degrees (circumferential axis as a reference) subendocardially to -60 degrees at the subepicardium. The counter coiled helix of subepi - and subendocardial fibres generates twist. The direction of basal and apical rotation is dominated by the larger - radius fibres at the subepicardium, caused by their longer arm of movement^[14]. The significance of the direction of fibres has been demonstrated in patient studies as well^[15]. Left ventricular twist showed a linear relation with sphericity index (as a measure of change in left ventricle fibre orientation, because in more spherically shaped hearts fibres are supposed to be oriented more horizontally) in patients with a dilated cardiomyopathy, supporting the hypothesis on twist mechanics and the influence of the direction of fibres on the twisting left ventricular deformation^[14].

The influence of aging on twist was studied by several groups^[16-19]. In all these studies, aging appeared to be related to an increase of left ventricular twist. As the function of the fibres at the subendocardium deteriorates when getting older, even in normal hearts^[20,21], the reduction of the opposing rotational forces of the subendocardium will result in an increase of apical rotation by the already dominant subepicardial fibres and consequently in an increase of left ventricular twist. This increase of left ventricular twist appears to be a part of “physiological cardiac aging”. One may hypothesize that this increase of twist contributes to the conservation of left ventricular stroke volume with ageing.

Untwisting begins after left ventricular twist reaches its peak, usually shortly before end-systole. Systolic twisting leads to storage of potential energy in the compressed coil of twisted fibres of the left

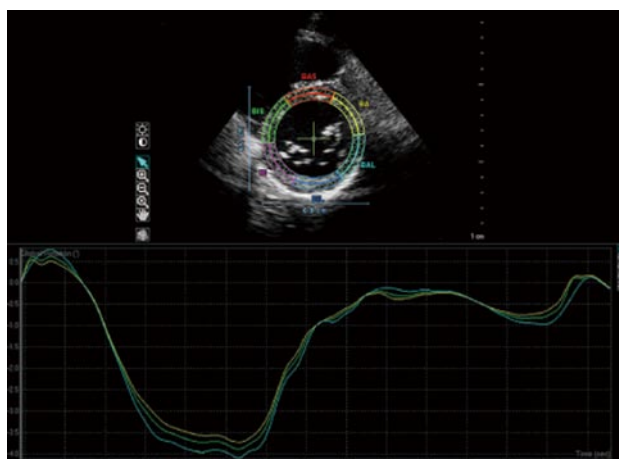


Figure 1 Example of QLAB workstation speckle-tracking analysis. Upper panel: 2D echo image of the basal left ventricular short axis. The software fully automatically draws the epicardial and endocardial contour of the myocardium. Lower panel: The software calculates the change in angle between the left ventricular (LV) wall and the virtual LV center during the cardiac cycle (green line). The blue and yellow line represent the angulation of the epicardial and endocardial angulation, respectively.

ventricular wall^[19]. During isovolumic relaxation this coil springs open and releases this energy. Fibres at the subepicardium that are still depolarized and are at that time - in contrast to systole - not overruled by active contraction of the fibres located at the subepicardium, might dynamically reinforce this role of untwisting in diastole^[19,22]. It was shown by magnetic resonance imaging that there is an important dissociation of time of untwist and filling, approximately 40% of the untwist takes place during isovolumic relaxation^[23]. Furthermore, the extent of the diastolic intraventricular pressure gradient is strongly related to untwisting. Even more so, untwist precedes the development of this pressure gradient, thereby potentially being an important indicator of suction during early diastole^[24,25].

TWIST MECHANICS IN CARDIOMYOPATHIES

Hypertrophic cardiomyopathy

Left ventricular twist in patients with hypertrophic cardiomyopathy is moderately increased, in particular the left ventricular basal rotation^[26-29]. This augmented rotation at mitral valve level is probably caused by reduced counteraction of the subendocardial fibres, due to subendocardial ischemia caused by endocardial microvascular insufficiency and increased oxygen demand^[30,31]. This is supported by the phenomenon of increased rotation being most pronounced in the hypertrophic segments^[26]. A larger difference in radius between the subepicardium and subendocardium will increase the arm of effect on the already dominant fibres of the subepicardium and consequently will increase rotation at mitral valve level (basal rotation)^[26].

There is a significant relation between the pattern of hypertrophy on apical rotation and twist. If the

septum has a sigmoid curvature, rotation of the apex is more pronounced than in reverse septal curvature hearts. Outflow tract obstruction is more common in patients in whom the septum has a sigmoid curvature. The resulting intraventricular forces from these outflow tract gradients can lead to microvascular insufficiency and thereby to more (sub)endocardial ischemia. Subsequently, this lack of oxygen might cause impairment of the countereffect of contraction of the subendocardially located myocardial fibres on left ventricular twist.

The necessity to objectively demonstrate diastolic dysfunction in hypertrophic cardiomyopathy has caused an ongoing pursuit for a non invasive and load independent technique for quantifying the severity of diastolic dysfunction. For instance Takeuchi *et al.*^[32] examined the effect of left ventricular hypertrophy in hypertensive patients on untwisting of the left ventricle. In moderate to severe hypertrophy, untwisting was reduced and delayed as compared to healthy individuals, supposedly resulting in decreased function of left ventricular diastole. In hypertrophic cardiomyopathy^[33] and also in aortic stenosis^[34] the untwisting rate, being the mean untwisting velocity during the isovolumic relaxation phase, is decreased and as a result untwisting is delayed^[27]. In hypertrophic cardiomyopathy this was most obvious in the affected segments^[27,29]. Also, compromised elastic characteristics lead to suboptimal transformation of the potential kinetic energy stored in the twisted heart. Peak diastolic untwisting velocity is reduced in hypertrophic cardiomyopathy whereas it is augmented in aortic stenosis. In aortic stenosis twist is increased more severe. Release of the relatively high amount of potential energy results in increased untwisting, possibly compensating for otherwise diastolic dysfunction^[27,35]. In hypertrophic cardiomyopathy twist is just discreetly increased, weakening this effect^[27,33].

Dilated cardiomyopathy

Twist in non-ischaemic dilated left ventricles is known to be reduced. The abnormal shape of the left ventricle in dilated cardiomyopathy may cause a change in fibre orientation. This fibre orientation is of importance in left ventricular twist as described earlier. This influence was found as an independent linear relation between left ventricular sphericity index and peak systolic twist. The more dilated the left ventricle, the more decreased the left ventricular twist. Actually, also in patients with dilated cardiomyopathy and comparable left ventricular ejection fraction, sphericity index was still significantly related to left ventricular twist^[15]. Nonetheless, derangement of myocardial fibre architecture is not the only cause for decreased left ventricular twist in patients with non-ischaemic dilated cardiomyopathy as fibrosis appeared to play a role in decreased left ventricular twist.

The extent of myocardial fibrosis in dilated cardiomyopathy has been evaluated by cardiac magnetic


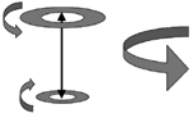
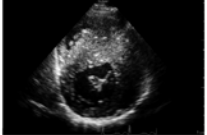
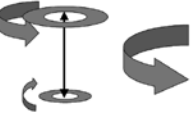
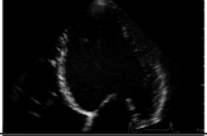
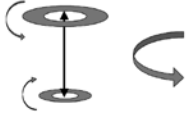
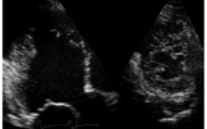
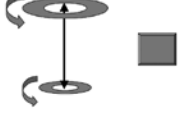
Overview of cardiomyopathies with corresponding abnormalities in twist mechanics			
Normal left ventricle			Normal basal clockwise rotation, normal apical counterclockwise rotation results in normal twist
Hypertrophic cardiomyopathy			Increased basal clockwise rotation, normal apical counterclockwise rotation results in increased twist
Dilated cardiomyopathy			Reduced basal clockwise rotation, reduced apical counterclockwise rotation results in reduced twist
Non compaction cardiomyopathy			Basal and apical rotation in the same direction results in rigid body rotation

Figure 2 Overview of cardiomyopathies with corresponding abnormalities in left ventricular twist mechanics.

resonance imaging with late gadolinium enhancement. Fibrosis proved to be related to twist^[36]. Reduction of twist indicated more extensive cardiac fibrosis.

As explained earlier, very rapid left ventricular untwisting is known to play a prominent part in fast filling in early diastole. However, in dilated hearts untwisting is delayed, leading to an apex-to-base-rotation delay. This will interfere with early diastolic suction and might harm filling of the left ventricle in dilated cardiomyopathy^[27,37,38].

Noncompaction cardiomyopathy

Noncompaction cardiomyopathy is still subject to debate because of the shortcoming on consensus on its pathogenesis, diagnosis and treatment^[39-41].

In the final embryonic development of the heart the myocardial tissue is transformed in a compact myocardium together with the formation of epicardial and endocardial fibre as oppositely wound helices^[6,42]. As noncompaction cardiomyopathy is supposed to be caused by intrauterine arrest of cardiac embryogenesis during this transformation^[43], distorted left ventricular twist features may be expected, even more than in the situation of reduced systolic function and a normally compacted myocardium. This was recognised in clinical studies^[44-48] where noncompaction cardiomyopathy patients displayed a twist pattern with basal and apical rotation in the same direction, resulting in almost full absence of left ventricular twist. This rotation pattern is known as left ventricular rigid body rotation^[44,49]. Rigid body rotation demonstrated, in a relative large study, to have a good predictive value for the diagnosis of noncompaction cardiomyopathy^[47]. Even more interesting, all familial noncompaction cardiomyopathy patients showed rigid body rotation. The fact that noncompaction cardiomyopathy diagnosis

is most definite in these patients underscores the good sensitivity of rigid body rotation in diagnosing noncompaction cardiomyopathy.

The clinical importance of left ventricular rigid body rotation was shown in more recent studies, where rigid body rotation was found in a majority of noncompaction cardiomyopathy patients as well^[50,51], but the patients with rigid body rotation and noncompaction cardiomyopathy proved to have a lower NYHA functional status as compared to the patients without rigid body rotation^[48].

Ischemic cardiomyopathy

An optical device attached to the apex was used in a canine model to study the early effects of myocardial ischemia^[52]. Ischemia was inflicted by occluding the anterior descending coronary artery. Early after induction of ischemia, there was a paradoxical increase of apical rotation. This finding was ascribed to secluded ischemia of the subendocardium, resulting in a declined counteractive effect of the fibres located at the subendocardium^[27,52].

Also, Moen *et al.*^[53] used speckle-tracking echocardiography on eight anesthetized pigs to define regional myocardial function in anterior wall ischemia. They discovered left ventricular twist remained normal until there was extensive impairment of perfusion of the left anterior descending artery.

Sun *et al.*^[54] induced a myocardial infarction in 7 pigs, leading to decreased twist, specifically in the area perfused by the occluded coronary artery^[27,54]. Hence, it was suggested that twist might be used to assess wall motion abnormalities in order to localize cardiac ischemia. Conversely, in a clinical study using dobutamine stress echo in 125 patients with myocardial infarction or ischemia, the influence of myocardial infarction on left ventricular twist proved to be related

to size rather than localisation of infarction. In addition, stress-induced myocardial ischemia did not influence left ventricular twist^[55]. Other studies on anterior myocardial infarction patients showed a decreased apical rotation in the infarcted left ventricle however with a preserved left ventricular basal rotation^[56,57].

When left ventricular myocardial infarction was complicated by left ventricular aneurysm formation, rotation of the apex was lost or even reversed, consequently losing left ventricular twist. Restorative surgery as a treatment of this problem is rather complex: the aim is to reconstruct a near normal ventricular chamber after aneurysm formation and thus reducing left ventricular volume and improving ejection fraction^[58]. Setser *et al.*^[59] did not see a significant improvement in their patients left ventricular twist after traditional left ventricular reconstruction. Much more interesting however; when an improved restoration technique was used, where residual myocardium around the defect was re-approached endeavouring to redirect fibre orientation displaced by infarct scar toward a more physiological gross disposition, left ventricular twist did improve in all patients^[60]. This encouraging concept of fibre orientation based surgical reparative surgery, could expand the potential of repairing the failing heart.

CONCLUSION

Left ventricular twist is an essential part of left ventricular function. Nevertheless, knowledge is limited in "the cardiology community" as it comes to twist mechanics.

Fortunately, evolution of echocardiography, permitting speckle tracking to precisely assess left ventricular twist, has boosted the awareness of this fundamental feature of cardiac mechanics. The vital role of twist in the physiology of the heart is undisputable. Nevertheless, the significance of twist assessment in daily clinical practice in patients with a cardiomyopathy still has to be established^[27]. On the other hand, twist analysis has contributed substantially to the understanding of pathophysiology in a diversity of cardiomyopathies (Figure 2). Increased twist in for example hypertrophic cardiomyopathy may be an early sign of subendocardial (microvascular) dysfunction. Furthermore, decreased twist might be initiated by left ventricular dilatation or an extensive myocardial scar. Finally, the detection of rigid body rotation in noncompaction cardiomyopathy could serve as an indispensable method to accurately diagnose this challenging entity. All this highlights the importance of left ventricular twist in the field of cardiomyopathies and may further encourage the implementation of left ventricular twist parameters in the "diagnostic toolbox" for cardiomyopathies^[27].

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