

## Left ventricular twist in patients with left bundle branch block: Missing the obvious in electromechanical coupling

The recent advent of imaging modalities capable of myocardial mechanical assessment, such as speckle tracking echocardiography, has offered insight for understanding the earlier and subclinical phases of cardiac dysfunction beyond the conventional parameters, such as ejection fraction (EF), and Doppler based methods (1, 2). However, myocardial functions are equally dependent on myocardial electrical properties because electromechanical coupling is crucial for an efficient cardiac contraction-relaxation cycle. One clear example of this concept is left bundle branch block (LBBB) that significantly influences left ventricular (LV)-electrical activation and contraction; the outcome is a dyssynchronous uncoordinated myocardial contraction that can progress to LV dilation and remodeling (3). In this issue, Yilmaz et al. (4) study the effects of LBBB on left ventricular twist (LVT), a crucial component of myocardial systolic and diastolic efficiency and the cross-road of both mechanical and electrical properties of the heart. To understand the significance of such an interesting question, a review of the mechanical and electrical concepts would be useful.

### LV rotational mechanics

The myocardium is a 3-dimensional continuum wherein the fibers change orientation gradually from an inner right-handed helix at the level of the subendocardium to an outer left-handed helix at the level of the subepicardium (5). The arrangement of fibers in such unique anatomical fashion leads to circumferential-longitudinal shear deformation that is visually perceived as rotation. However, this opposing counter-directional arrangement would intuitively make both layers rotate in opposite directions such that the contraction of subepicardial fibers would rotate the apex counter-clockwise and the base clockwise, whereas the contraction of subendocardial fibers would rotate the apex clockwise and base counter-clockwise. However, these two counter-directional rotational movements do not cancel out because of the longer radius of the outer epicardial layer that generates a larger lever arm force leading to the domination of the overall direction of rotation over the subendocardial fibers with the resultant final rotational outcome that is counter-clockwise at the apex and clockwise at the base (the directions of the subepicardial fibers) (5). Another important observation is that, because of the geometrical nature of the helix, an opposing api-

co-basal gradient of rotation always exists and is defined as LVT, a motion that resembles the wringing of a cloth to squeeze out water. With the onset of diastole, the stored energy in the myocardial wall created by LVT is utilized for diastolic recoil when it is released during early relaxation [untwist (UT)] generating diastolic suction pressure (5).

Because LVT and UT characterize LV systole and diastole, respectively, parameters describing these mechanical behaviors can be useful in the assessment of LV systolic and diastolic functions. Examples of such parameters include the magnitude of LVT at peak systole (measured as the difference between basal and apical rotation in degrees) and the percentage of LV-UT at early diastole as well as the early diastolic LV-UT rate (in degrees/second) that can reflect diastolic LV relaxation and its expected hemodynamic significance.

### Electromechanical coupling in the light of LV rotational mechanics

Myocardial electrical activation is not transmurally homogeneous. With the onset of excitation at the upstroke of the electrocardiographic R wave, the depolarization wave travels to the LV septal subendocardial fibers first; therefore, septal subendocardial segments are first to be excited from apex to base, while the basal posterior wall is the last to be activated during the downslope of the R wave. This timing sequence of electrical excitation is influenced by the impulse propagation through the His-Purkinje system and the anisotropic nature of myocardium that facilitates conduction along rather than across the fibers. Repolarization, on the other hand, occurs in the opposite sequence, as it propagates from epicardium to endocardium and from base to apex. Thus, the apical subendocardium, which was the first region to undergo depolarization will be the last region to complete repolarization (6).

The rapid apico-basal spread of electrical activation during depolarization initiates early contraction at the septal subendocardium causing shortening during the isovolumic contraction. Subendocardial shortening is accompanied by simultaneous subepicardial fiber stretching, which retains the LV cavity within isovolumic constraint (i.e. shortening in one direction is cancelled out by stretching in the other direction). The transmural spread of activation eventually reaches to the subepicardial fiber causing their

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contraction, which coincides with the onset of systolic ejection.

Here, it is important to note that subendocardial shortening and subepicardial stretching contribute to a brief clockwise rotation of LV apex because, at this time, the active part of the myocardium is the subendocardium at the apex and mid-septum, and thus, the opposing force of the subepicardium is lacking, leading to a rotation force that follows the endocardial directions (i.e. clockwise at the apex and counter-clockwise at the base), which visually resembles the directions during diastolic UT (6).

### Effects of LBBB on electromechanical coupling and LV rotation

LBBB has a complex influence on the process of LV electric activation and contraction, resulting in mechanical dyssynchrony that causes LV dilation, remodeling, and the progression of LV systolic and diastolic dysfunction. The loss of coordinated myocardial contraction in LBBB patients correlates with the depression in LV systolic function irrespective of the cause of LBBB. The internal loss of the early septal excitation in LBBB also results in the redistribution of mechanical activity, leading to the disruption of the electromechanical coupling. Intra-ventricular asynchrony created by LBBB alters the sequence and duration of myocardial depolarization; thus, the redistribution of the global mechanical activity and the delay of the septal excitation result in the disruption of the sequence of LV rotation mechanics. However, the clinical impact on the development of LV dilation and the progression to overt heart failure in these patients is understudied.

"Left ventricular twist was decreased in isolated left bundle branch block with preserved ejection fraction." published in this issue of *Anatol J Cardiol* 2017; 17: 475-81. Yilmaz et al. (4) have shown that the presence of LBBB in patients with preserved EF will alter myocardial rotational mechanics and may be a representation of the subtle systolic dysfunction. In their study, the authors noticed that parameters of diastolic function are also significantly affected in these patients. Given the role of LV diastolic UT in the generation and maintenance of effective diastolic functions, it would have been of greater value to study the relationship of the parameters of UT to the development of diastolic dysfunction in patients with LBBB and to compare them to diastolic dysfunction in patients without LBBB. Indexing LVT to some hemodynamic parameters like the ratio between early diastolic mitral flow velocity to early diastolic mitral annular velocity ( $E/e'$ ), would also be interesting to consider and may provide some deeper insights into the link between systolic and diastolic dysfunction in such patients. Finally, and more importantly, given the regional nature of the disease, it would have also been of great value if one could assess regional rather than global twist and enrich these results with parameters of segmental time to peak twist and early diastolic UT to elucidate the mechanisms

underlying the dysfunction as an outcome of electromechanical dissociation in these patients.

### Conclusions and future directions

LVT is an excellent example of the vital role of electromechanical coupling for myocardial efficiency. LBBB introduces severe incoordination in myocardial electromechanical coupling that can be noticed at a subclinical level and becomes more pronounced with LV dilation and heart failure development. Such relationships are closely related to the disruption of the sequence of LV rotation mechanics, which may contribute to LV dysfunction. Future studies, besides focusing on the relationships between LVT–UT and electromechanical coupling in patients with LBBB, should also look into how such complex pathological relationships may influence the intraventricular flow dynamics and LV vortex formations, another important part of an efficient LV pumping (2). These regional electrophysiological, electromechanical, and hemodynamic observations can help to differentiate causes of cardiomyopathy associated with LBBB and may also add to the understanding and selection for cardiac resynchronization therapy with a better responder outcome.

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