Letter by Dwivedi et al Regarding Article, “Left Ventricular Untwisting Rate by Speckle Tracking Echocardiography”

To the Editor:

We read with interest the article by Wang et al1 that compared left ventricular (LV) twist, twisting rate, and untwisting rate (UR) in subjects with LV systolic dysfunction and depressed ejection fraction (EF), subjects with diastolic dysfunction and normal EF, and 20 healthy subjects by speckle tracking echocardiography. This study highlights major issues in LV diastolic function. However, we believe that it contains some limitations that could modify interpretation of the conclusions.

The main result of the clinical study is that LV twist, twisting rate, and UR are reduced in patients with LV systolic dysfunction and depressed EF but not in those with diastolic dysfunction and normal EF. The finding of normal UR in patients with diastolic dysfunction and markedly reduced mitral annular Ea velocities implying reduced recoil is divergent from a number of other studies,2,3 which have shown that patients with diastolic dysfunction have increased resting torsion but prolonged untwisting and delayed peak untwisting, even at rest. Indeed, a recent study has shown that peak UR was an independent predictor of τ, the relaxation time constant.4 The authors justified their finding of preserved UR in diastolic dysfunction by quoting a study where subjects with hypertrophic cardiomyopathy and diastolic dysfunction were found to have nonsignificantly different albeit low resting untwisting velocity (1.6±0.8 versus −2.0±0.7 rad/s, P=0.2), compared with normal subjects.4 But what is not mentioned is that in the same study this difference in velocity became more obvious and significantly different with exercise. Furthermore, it is worthwhile to note that although peak systolic torsion was greater than that of normal subjects at rest (in agreement with the study by Wang et al), it did not augment with exercise and also it was less efficient at generating untwisting both at rest and during exercise.

One of the explanations given in the article for preserved UR in patients with diastolic dysfunction is a normal or reduced end-systolic volume in this group and higher ratio of N2B to N2BA titin isoforms. We believe that this reasoning is too simplistic. Transmural gradients and differences in orientation of muscle fibers and cleavage planes may be more important and may lead to paradoxical effects: reduced subendocardial fiber action at the apex may lead to increased twist because of unopposed action of the subepicardial left-handed helical fibers. Another source of the restoring force that drives diastolic torsional recoil is systolic deformation within the sheet structure, because during systole significant shearing of the fibers within the sheet plane occurs.5

In summary, the authors are to be congratulated for having performed an interesting study but, given the limitations, we should be cautious about their conclusions until larger studies can be performed using different techniques and especially including exercise.

Disclosures

None.

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References

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