About left ventricular torsion, sex differences, shear strain, and diastolic heart failure

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This editorial refers to ‘Preserved left ventricular twist and circumferential deformation, but depressed longitudinal and radial deformation in patients with diastolic heart failure’† by J. Wang et al., on page 1283

Wang et al.,† provide novel pathophysiological information on patients with diastolic heart failure, by exploring the fourth dimension of cardiac function: left ventricular (LV) twist. The complex active and passive physiological events occurring during the heart cycle are related to the complex structure of the heart, which is a helix that contains an apex. Lower (17th century) described the cardiac helix form as having an apical vortex, in which the muscle fibres go from outside in, in a clockwise way, and from inside out, in a counter-clockwise direction.2 Nature contains many pathways of clockwise and counter-clockwise spirals (reciprocal spirals), e.g. the flower buds of a daisy, the sea shell, and even human fingertips. Dr Torrent-Guasp compared the structure of the heart with a coiled rope with a beginning and an end at the aorta and pulmonary artery; a wraparound loop called a basal loop; and a helix that he called the apical loop.3 Does this ventricular architecture correspond to a functional reality? The classical view relates to contracting and relaxing, as defined by William Harvey (1628): constricting to narrow and eject, dilating to widen and fill. In a comprehensive review, Dr Buckberg describes that the heart cycle is not merely constricting and dilating but that it comprises four fundamental motions that include narrowing, shortening, lengthening, and widening.2 The initial contraction involves the basal loop that causes constriction whereby a stiff outer shell muscle is formed that surrounds both ventricles. This corresponds to the isovolumetric, pre-ejection phase of systole. The next sequence is the contraction of the descending loop, which twists downward to thicken and cause ejection. Contraction of the ascending segments starts shortly after the descending segment and persists after the descending segment contraction stops,4 producing untwisting (isovolumetric relaxation) and creating suction (early filling). Finally, relaxation is completed, and passive filling of the ventricles and atrial contraction occur. As viewed from the LV apex, the early-systolic shortening of subepicardial fibres results in a counter-clockwise rotation of the LV apex and a clockwise rotation of the LV base. The net result is a twist that results in a wringing movement of the LV.5 During isovolumetric relaxation, the twisted subendocardial fibres behave like a compressed coil that springs open while releasing the potential energy stored in the deformed matrix. This recoil is appreciated from the apex as a clockwise rotation of the LV apex.

In the case of heart failure with diminished ejection fraction, the helical formation of the heart is replaced by a spherical chamber causing a diminished capacity to shorten and lengthen. When heart muscle fibres adopt a spiral formation, proceeding toward the apex, 15% fibre shortening causes a 60% LV ejection fraction. If fibre orientation is horizontal or transverse, the same 15% fibre shortening produces only a 30% ejection fraction.6 The underlying anatomy of heart failure with diminished LV ejection fraction may reflect changing the oblique apical loop into a more transverse basal loop architecture, with resultant diminished function.

Until now, the lack of easily applicable non-invasive techniques to study the LV myofibre architecture in relation to the spatiotemporal sequence of regional deformations occurring during cardiac contraction and relaxation has prohibited extensive in vivo experiments, especially in patients with diastolic heart failure. A recently introduced echocardiographic technique—speckle tracking—enables non-invasive evaluation of these complex cardiac events. Speckle tracking or two-dimensional strain is a technique that tracks frame-to-frame movement of natural acoustic markers, or speckles, present on standard two-dimensional ultrasound tissue images.7 Local two-dimensional tissue velocity vectors are derived from the spatial and temporal data of each speckle. Myocardial strain can be assessed from temporal differences in the mutual distance of neighbouring speckles. From short axis images, circumferential and radial strain can be calculated and,
from apical images, longitudinal strain can be derived. This allows non-invasive evaluation of longitudinal shortening, radial thickening, and circumferential shortening of myocardial muscle. Moreover, speckle tracking was also validated for the measurement of cardiac rotation and twist. It is simple and reproducible: it only requires two-dimensional acquisition of parasternal short axis images at the basal level and the apical level. Apical and basal rotation is measured from frame-to-frame tracking of grey-scale speckle patterns. LV twist is calculated as the difference in rotation between the apex and the basal segment. LV torsion and twist are not interchangeable, as torsion represents twist normalized for distance.

In their study, Wang et al. showed that in patients with systolic heart failure, longitudinal, circumferential, and radial strain, and LV twist all are impaired. In contrast, in patients with diastolic heart failure, LV longitudinal and radial strains are reduced, but circumferential strain and LV twist are preserved. LV ejection fraction and circumferential strain are the independent determinants of LV twist. The study was performed on a limited number of consecutive patients. They were not perfectly matched for age, gender, and disease severity. The preliminary character of the report was acknowledged in the manuscript.

It is common practice to subdivide patient with heart failure into those with systolic and diastolic heart failure, according to the ejection fraction. In systolic heart failure, the heart works by way of a subtle balance between a steep diastolic and a flat, horizontal systolic pressure–volume relationship. The stroke volume is limited by the inability to decrease systolic volume under workable blood pressures. The predominant abnormality of myocardial cell contraction and the remodelling lead from the start to loss of torsion, i.e. loss of the wringing motion, as explained above. In diastolic heart failure, the heart works by way of a subtle balance between a steep diastolic and an even steeper systolic pressure–volume relationship (Figure 1). Stroke volume is limited by the inability to increase diastolic volume and to decrease systolic volume further. In early diastolic heart failure, torsion may be regarded as a compensatory mechanism to combined and balanced contraction and relaxation abnormalities of the myocardial cell.

This compensation contributes to the preservation of the ejection fraction. For example, pressure overload induced by aortic stenosis is associated with an increase of systolic LV wringing motion. This mechanism, however, declines with increasing LV hypertrophy and dilatation. A similar compensation was observed in diabetic type I patients. In the contribution under scrutiny, torsion was as a mean normal. This could suggest more advanced disease than in the former two studies, heart failure with pseudonormalization of torsion. It remains to be investigated how this pseudonormalization differs from normal physiological torsion.

In addition to quantitative aspects of twist, the rate and timing of twisting and untwisting should be further analysed. The untwisting of the heart predominantly occurs during isovolumetric relaxation. Preservation or impairment of torsion during isovolumetric relaxation can add to our insight on diastolic heart failure. In patients with hypertension and hypertrophy, diastolic untwisting is delayed and reduced, but in diastolic heart failure untwisting is preserved. Such discrepancies should be clarified.

We might speculate why circumferential strain parallels to some extent torsion in diastolic dysfunction, while longitudinal function is depressed. Torsion is a contributor to circumferential strain in addition to myocardial fibre shortening. However, in the model of Torrent-Guasp, torsion should be affected by longitudinal function as well, unless the downward systolic motion of the cardiac base can be uncoupled from the torsion: in hypertrophy and diastolic dysfunction, combined torsion and longitudinal function have evolved toward even increased torsion and loss of longitudinal function. This paradox should primarily be investigated with the analysis of shear strains, but also in terms of the velocity, duration, and extent of longitudinal shortening, and in terms of re-orientation of myocardial fibres in hypertrophy.

Further research should describe the contribution of torsion and three-dimensional strains to the dysfunction in various stages and aetiologies of cardiac failure. Gender differences should be actively searched for. While women tend to respond to overload and injury by hypertrophy and fibrosis, men tend to respond more by contractile dysfunction and dilatation. The underlying mechanisms of sex-related differences and hormonal effects on cardiac remodelling remain poorly investigated, but could be related to differential constitutive nitric oxide synthase regulation and its effects on the myocardium and coronary vasculature. This could manifest at early stages of the response to load or injury as differential behaviour of torsion between men and women.

Finally, in the way that clinical researchers think and if possible in their computations, they should not forget the importance of shear strains in addition to three-dimensional strains. Only then will they be able to understand total deformation and function. Computation of longitudinal/circumferential shear is another promising approach to the assessment of torsion with speckle tracking, allowing in addition the investigation of regional differences in torsion.

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References

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