LETTERS TO THE EDITOR

Reduced and delayed untwisting of the left ventricle in patients with hypertension and left ventricular hypertrophy: a study using two-dimensional speckle tracking imaging

We read with interest the study by Takeuchi et al.1 which elegantly lends weight to the critical role of untwisting in normal and abnormal diastolic function.2 However, we would like to raise two important methodological issues in the use of speckle tracking imaging (STI) to assess torsion:

(1) The need for further interpolation of STI data. The STI algorithm includes cubic spline interpolation and provides estimation of rotation in the six predefined segments at each frame. Because current 2D echocardiography allows imaging only in one plane at a time, changes between acquisition of the apical and basal short-axis images in heart rate or frame rate, as documented by these authors, may result in the STI algorithm estimating rotation for frames at different time points at the apex and base. Although temporal normalization overcomes intersubject differences in heart rate, it does not prevent deduction of basal rotation from apical rotation values at different time points in the cardiac cycle which results in erroneous calculation of torsion or twist. A further cubic spline interpolation1,3 of the temporally normalized apical and basal rotation data overcomes this issue by ensuring data from the same time points can be deducted. Although this is potentially labour intensive, customized automated algorithms may facilitate this.4

(2) The measurement of untwisting and untwisting rate from end systole. Because peak torsion, which is the onset of untwisting, may be delayed beyond aortic valve closure, particularly, as these authors and others have shown, by increased LV mass,3 we believe that the untwisting should be measured from peak torsion rather than end systole. Measurement of untwisting from end systole may result in a significant underestimation of the gradient of untwisting (e.g. in this study, Table 3, untwisting at t = 5%, for the severe left ventricular hypertrophy group, a negative value does not represent true untwisting). Both timing and velocity of untwisting may be important in diastolic dysfunction and the parameter negative torsion acceleration5 incorporates both these aspects of torsional dynamics and may be a useful parameter of diastolic function. In the present study, because the authors have calculated untwisting as a percentage, the units of untwisting rate should be percentage per millisecond.

We commend the authors on this excellent work but emphasize the importance of consistent and reproducible methodology for the ongoing study of left ventricular torsion.

References


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We thank Dr Burns for the interest in our work1 and on the appropriate comments about the methodological issues in the use of 2D speckle tracking imaging (STI) to assess left ventricular (LV) twist, i.e. interpolation of STI data and measurement of untwisting.1,2 Left ventricular twist has been defined as apical rotation—basal rotation,1,3,4 if time-

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domain analysis is required, bottom line is that frame rate and heart rate in both views should be the same. To accomplish this, we always acquired both level of images with the same depth and the sector size to ensure frame rate is identical. In addition, we are very keen about the heart rate when we acquired images. If the differences of heart rate from both level were than >5 b.p.m., we tried to obtain another image to reduce the difference in heart rate. Because held respiration during image acquisition often makes heart rate slow, it is not always easy to obtain the same heart rate in both images. However, minor differences in heart rate between the two images produce only two or three frames more in one view. This does not tremendously affect the calculation of LV twist, because the number of frames during systole is usually the same, and only differences would be occurred in the later part of diastole, which does not affect our calculation of untwisting and untwisting rate. Although Dr. Burns claimed cubic spline interpolation of the temporally normalized apical and basal rotation data might overcome this issue, careful acquisition of images could alleviate this problem. Left ventricular untwisting, which is thought to contribute diastolic LV suction, is predominately observed during isovolumic relaxation period in normal heart. Thus, untwisting should be evaluated from aortic valve closure to early diastolic phase. A negative value of untwisting at $t = 5%$ in our study (Table 3) reflects the presence and severity of delayed onset of untwisting after the aortic valve closure, and it also suggests that LV still continuously twists for the first 5% of diastolic period, resulting in the adverse impact on LV early diastolic filling. Finally, we apologize the unit of untwisting is not degree/ms but %/ms as the authors suggested. Although 2D STI assessment of LV untwisting is a promising method for providing new indices in the evaluation of early diastolic function, further technological refinements and more user-friendly software is mandatory to expand this technology in the daily clinical practice.

References


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As one of the cohorts for recent genome-wide association studies, we have experienced firsthand the difficulty with accurate phenotyping, especially for CAD. Atherosclerosis is a diffuse process, and coronary angiography may not always reveal the true burden of disease, especially if the vessel has undergone Glagov remodelling. The authors comment on and analyse LMD as a discrete entity, but clinical experience suggests that LMD is rarely an isolated phenomenon. More commonly, LMD occurs with significant CAD elsewhere in the coronary circulation. In our genetic registry of all patients passing through the catheter laboratory, we have over 2000 coronary angiograms available for analysis. Of the patients who had an MI, we found 64 patients with LMD >50% diameter stenosis. Of these patients, those with LMD, in addition to one-vessel disease (VD), two-VD, and three-VD were 22%, 28%, and 45%, respectively (unpublished data). Only 5% had LMD alone. Fischer et al. do not address this distinction and in the absence of that data, we would argue that the heritability of LMD is actually a surrogate of heritability of severe CAD as manifested by the number of vessels involved. This would also explain the findings that relatives of LMD patients have a higher risk of developing an event, as they would have inherited a more severe underlying atherosclerotic process. This paper raises important questions about the inheritance of CAD. The notion that disease is, in part, genetically driven to occur primarily in certain sites in families defies current evidence and our current understanding of the atherosclerotic process. Alternative explanations include the role of flow mechanics in the development of lesions especially in the proximal coronary tree. We know from an expanding evidence base that low-wall shear stress (WSS) contributes to plaque development in anatomically predisposed sites. It seems plausible then, that low WSS in anatomically identical vascular regions would lead to plaque development in the same sites. However, coronary anatomy is rarely identical, even in monozygotic twins and so this theory also fails to fully explain the rationale for site-specific coronary lesion inheritance.

In conclusion, we congratulate the authors on their important attempt to narrow the phenotype of coronary disease in order to make genetic studies more meaningful. However, we would argue from a pathophysiological perspective that a hereditary basis is more likely to explain underlying processes leading to atherosclerosis rather than a site-driven mechanism, which is difficult to link.