Quantitative Assessment of Intrinsic Regional Myocardial Deformation by Doppler Strain Rate Echocardiography in Humans

To the Editor:

We have read with interest the recent report of Edvardsen et al in which left ventricular regional peak systolic strain values measured by ultrasound were correlated with the corresponding strain data extracted by 3-dimensional tagged MRI. The study showed an overall acceptable agreement between the 2 techniques for several myocardial segments in both normal and pathological myocardium and for both radial and longitudinal deformation.

Although the longitudinal deformation parameters measured were in agreement with prior results, the radial values were not. Indeed, in this study, Lagrangian radial strain in the posterior wall of healthy volunteers was measured to be 18±9% and 17±6% by ultrasound and MRI, respectively. However, previously reported studies using standard M-mode echocardiography had found radial maximal systolic Lagrangian strain values of 69±23%.

The discrepancy between these values and the ones published in the study of Edvardsen et al could, in theory, be attributed to the fact that the authors used newer techniques based on MRI tagging and Doppler myocardial imaging, and that these techniques might be more accurate. However, previous cardiac MRI studies, both with and without tagging, had already shown Lagrangian radial strain in the posterior wall to be around 33±9% and 37±9%, respectively. Moreover, using Doppler myocardial imaging based strain estimation, values around 60±13% have been reported. Although a small decrease in radial strain with aging has been described, it cannot account for the large discrepancy with the values found in the study by Edvardsen et al.

At least 4 independent studies using 4 different techniques have thus reported Lagrangian radial strain values of the posterior wall to be within the range of 35% to 60% for normal individuals around 40 years old. The values published in the paper by Edvardsen et al thus seem to be too low to be realistic for radial deformation in normal subjects. This might suggest that there is either a consistent methodological error in radial strain estimation in their paper (this is unlikely as both techniques gave similar values) or that the myocardial segment analyzed was too close to the fibrous mitral ring, as this region is known to deform less.

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Response

In their letter, D’hooge et al conclude that the radial systolic strain values reported in our article seem too low to be realistic for normal left ventricles. They base this conclusion on comparison with previous reports using different methodologies.

When D’hooge et al refer to Lagrangian strain values of 63±23% by M-mode echocardiography, they probably mean wall thickening and not strain. Wall thickening measured by M-mode echo and MRI without tissue tagging is in part due to systolic folding of the endocardium, whereby trabeculae join and become included within the left ventricular (LV) wall. The confluence of trabeculae may be considered an increase in wall thickness, but that does not represent strain. Therefore, the difference between wall thickening by M-mode echo and strain Doppler in our study may not be a matter of accuracy, as suggested by D’hooge et al; it may reflect different measurement principles.

As noted by D’hooge et al, however, the strain values found by tissue tagging and Doppler echocardiography in some previous studies exceed those measured in our study. The measurement of radial LV strain, however, is complicated by a large transmural strain gradient. In a theoretical model, Hexeberg et al calculated that systolic radial wall thickening (which in this theoretical model was equivalent to strain) was 52%, 27%, and 18% in the inner, mid, and outer myocardial layers, respectively. In vivo observations of circumferential strain confirm a large transmural gradient. One may speculate that some of the previous studies have measured strains in layers with the highest value. We showed consistency between 2 different techniques that measured strains over a region that probably included most of the LV wall thickness. The measurements were not taken close to the mitral ring, as suggested by D’hooge et al.

When measuring radial strain by Doppler the sample volume moves continuously across the LV wall and may also include parts of the ventricular cavity and extracardiac space because of cardiac translation. This may be one reason why radial strains have more variability than longitudinal strains, with standard deviations of ±6 to 9% and ±3 to 4%, respectively.

Thus, there are several reasons why radial strain values may be technique-dependent, and it is unlikely that there is a radial strain value that is unique for the myocardium. It does appear that longitudinal myocardial strains are more robust measures of cardiac function than radial strains.

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