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**EDITORIAL COMMENT**

# Speckle-Derived Strain

## A Better Tool for Quantification of Stress Echocardiography?*

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Detection of chronic coronary artery disease (CAD) via visual assessment of wall motion abnormality is subjective and variable (1). Tissue Doppler echocardiography (TDE) and tissue Doppler-based strain echocardiography (TDSE) allow quantification of regional myocardial mechanics (2,3). The concept of using regional mechanics for stress echocardiography is appealing because rest or inducible ischemia ultimately manifests as abnormal regional mechanics.

Tissue Doppler echocardiography and TDSE have offered several options for quantifying inducible ischemia (4,5). Quantification appears to be superior to traditional visual assessment of wall motion and may help novice echocardiographers in interpretation of stress echocardiograms (6). The primary parameter used to track changes in regional mechanical behavior is systolic displacement (tissue velocity) or deformation (systolic strain rate or strain).

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A major limitation of current TDE and TDSE is that peak amplitudes, and to some extent phase (timing), of velocity and strain variables are influenced by the angle of the incident ultrasound beam with the myocardial wall (7). This restricts imaging to the apical projections wherein the operator attempts to align the myocardial wall parallel to the ultrasound beam; however, this is not always possible. Angle issues may be circumvented by narrowing the sector and imaging single walls. Image collection is duplicated as TDE images are collected in addition to standard B-mode images. These issues make TDE challenging and time consuming, thus reducing its wider applicability. Lastly, the complex 3-dimensional deformation of the heart during the cardiac cycle may not be adequately captured by investigating velocity and strain only in the longitudinal direction. This limitation may or may not impact the ability of this method to accurately detect inducible ischemia.

Newer speckle-tracking methods, which are not Doppler based, are not influenced by the angle of the incident beam (8,9). Thus, they can be potentially used in any projection and without paying much attention to the orientation of the heart in the imaging sector. A speckle is a unique acoustic pattern resulting from the interaction of ultrasound energy with tissue. These unique patterns can be tracked automatically over periods of the cardiac cycle, thus providing information about motion and displacement of that particular region of the myocardium. This information is used to derive strain and strain rate. Speckle-derived strain has obvious advantages in stress imaging. B-mode images can be collected as in usual clinical practice; therefore, there is no duplication of image acquisition. Although this method is unreliable right now, there is the theoretic possibility of simultaneously measuring strain in more than 1 direction, unlike TDE/TDSE.

Although speckle-derived strain has been validated in various circumstances, there is a paucity of data supporting its use in stress echocardiography. In that regard, the paper by Reant et al. (10) in this issue of the *Journal* provides important new information on the potential feasibility and utility of speckle-derived strain in stress evaluation. Because speckle-derived strain uses images at much slower frame rates (40 to 90 frames/s) than TDE (>100 frames/s), there is a concern that strain rates may not be as accurate at the faster heart rates associated with stress.

Using an open-chest animal model, Reant et al. (10) examined the validity of strain measurements under varying conditions of flow-limiting and nonflow-limiting coronary artery stenosis at rest and during dobutamine infusion using an ultrasonic flow probe around the coronary artery to gauge the level of stenosis. These investigators examined strains in more than 1 direction, thus providing the relative accuracy of radial, circumferential, and longitudinal strains for the diagnosis of inducible ischemia. Myocardial deformation by sonomicrometric crystals was used as the reference standard for strain. Correlation and agreement between speckle-derived strain and sonomicrometry was best for longitudinal strain (at rest and during dobutamine), modest for circumferential strain at rest and better during the dobutamine infusion, and modest for radial strain at rest and during the dobutamine infusion. Invasive hemodynamic measurements indicated no differences in global function among the various states. Interestingly, longitudinal and circumferential strains were abnormal at rest during flow-limiting stenosis and during stress with nonflow-limiting stenosis, whereas radial strain was only abnormal during stress in the setting of flow-limiting stenosis. This suggests that strains in the longitudinal and circumferential directions may be

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more sensitive to changes in regional deformation than radial strain.

Reant et al. (10) add to the growing body of evidence supporting the use of deformation analysis for detection of inducible ischemia (5,11,12). However, the results have to be interpreted in the context of certain limitations, some of which are acknowledged by the authors. An open-chest model alters regional mechanics. Although the article establishes proof of principle, it is unclear if the character and magnitude of change will be similar in clinical practice. The authors measure flow but not regional perfusion. The 2 are closely related but not the same. Nonetheless, their results are generally concordant with closed-chest models of dobutamine stress that measured regional perfusion and demonstrated similar changes in strain, albeit with Doppler-derived methods (13). There are no data on wall motion changes, and thus it is unclear whether changes in strain preceded or were more sensitive than changes in wall motion, as indicated in previous experimental models (14). There are no data on post-systolic strain, which has been demonstrated to be of value in detecting inducible ischemia (5,15).

Reant et al. (10) reiterate the potential limitations of radial strain noted in previous studies (16,17). However, the reasons why radial strain may be unreliable remain unclear. Is radial strain truly not as sensitive to induced ischemia or are these differences related to technical issues? Radial strain measurement does not correlate well with sonomicrometry, demonstrates larger variability, and has been measured in fewer locations compared with longitudinal strain. Poorer lateral resolution may contribute to less accurate calculations of radial strain. Any or all of these factors could underlie the lack of sensitivity of radial strain. Lack of sensitivity may only reflect the lack of a suitable method to accurately measure it. An attractive alternative explanation is that the longitudinal fibers located in the subendocardium, thus mediating long axis deformation, are more susceptible to ischemia and are therefore affected earlier in the ischemic cascade (18).

The accuracy of speckle-derived strain measurements at faster heart rates is an issue. Intuitively, one would expect the accuracy of measurements to be worse during faster heart rates because less frames would be recorded per cardiac cycle compared with resting heart rates. However, the authors note that interobserver and intraobserver variabilities were lower during dobutamine infusion. This finding may be related to a reduced dynamic range of strain measurements during faster heart rates rather than a true reduction in variability.

Finally, what does this article portend for the clinician performing stress echocardiography? In our experience, measurements of speckle-derived strain measurements in the clinical population remain fairly variable at rest and with stress, especially with radial strain. The finding that longitudinal strain is more sensitive is reassuring only in the sense that we will need to rely less on the highly variable radial strain. However, if we are going to use longitudinal strain to recognize inducible ischemia, is there a compelling reason to use speckle-derived strain versus TDSE? As alluded to earlier, TDSE offers the opportunity to acquire images at very fast frame rates (approximately 250 frames/s). Do the theoretic advantages of speckle-derived strain outweigh its limitations, and more importantly, will they provide incremental and superior diagnostic capabilities over fast frame rate TDSE?

Fast frame rate TDSE offers a high-fidelity signal for assessment of systolic and diastolic strain rates that is not available with speckle-derived methods. Changes in systolic or diastolic strain rate may be useful in detecting inducible ischemia. Diastolic strain rates may be complementary to and more specific than systolic strain rates for the diagnosis of ischemia (19). Speckle-derived methods appear to yield superior strain but not strain rate data. Whether this is a reasonable trade-off in the overall evaluation of CAD is unclear at this time.

To summarize, Reant et al. (10) provide a systematic validation of speckle-derived strain during dobutamine stress and indicate that longitudinal or circumferential strain is more sensitive than radial strain. They confirm the large variability and the resulting unreliability of radial strain. This observation may warrant cautious interpretation of radial strain data. The growing evidence in favor of deformation analysis for the detection of inducible ischemia highlights the pressing need for better instrumentation and analysis programs for reliable strain analysis.

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## REFERENCES

1. Hoffman R, Lethen H, Marwick T, et al. Analysis of interinstitutional observer agreement in interpretation of dobutamine stress echocardiograms. J Am Coll Cardiol 1996;27:330–6.
2. McDicken WN, Sutherland GR, Moran CM, Gordon LN. Colour Doppler velocity imaging of the myocardium. Ultrasound Med Biol 1992;18:651–4.
3. Sutherland GR, Di Salvo G, Claus P, D'Hooge J, Bijnens B. Strain and strain rate imaging: a new clinical approach to quantifying regional myocardial function. J Am Soc Echocardiogr 2004;17:788–802.
4. Madler CF, Payne N, Wilkenshoff U, et al. Non-invasive diagnosis of coronary artery disease by quantitative stress echocardiography: optimal diagnostic models using off-line tissue Doppler in the MYDISE study. Eur Heart J 2003;24:1584–94.
5. Voigt JU, Exner B, Schmiedehausen K, et al. Strain-rate imaging during dobutamine stress echocardiography provides objective evidence of inducible ischemia. Circulation 2003;107:2120–6.
6. Fathi R, Cain P, Nakatani S, Yu HC, Marwick TH. Effect of tissue Doppler on the accuracy of novice and expert interpreters of dobutamine echocardiography. Am J Cardiol 2001;88:400–5.
7. Heimdal A, Abraham T, Pislaru C, Belohlavek M. Angle dependency of strain rate imaging in an animal model. J Am Soc Echocardiogr 2000;13 Suppl:484.
8. Helle-Valle T, Crosby J, Edvardsen T, et al. New noninvasive method for assessment of left ventricular rotation: speckle tracking echocardiography. Circulation 2005;112:3149–56.

9. Reisner SA, Lysyansky P, Agmon Y, Mutlak D, Lessick J, Friedman Z. Global longitudinal strain: a novel index of left ventricular systolic function. J Am Soc Echocardiogr 2004;17:630–3.
10. Reant P, Labrousse L, Lafitte S, et al. Experimental validation of circumferential, longitudinal, and radial 2-dimensional strain during dobutamine stress echocardiography in ischemic conditions. J Am Coll Cardiol 2008;51:149–57.
11. Hanekom L, Cho GY, Leano R, Jeffriess L, Marwick TH. Comparison of two-dimensional speckle and tissue Doppler strain measurement during dobutamine stress echocardiography: an angiographic correlation. Eur Heart J 2007;28:1765–72.
12. Hanekom L, Jenkins C, Jeffries L, et al. Incremental value of strain rate analysis as an adjunct to wall-motion scoring for assessment of myocardial viability by dobutamine echocardiography: a follow-up study after revascularization. Circulation 2005;112:3892–900.
13. Yip G, Khandheria B, Belohlavek M, et al. Strain echocardiography tracks dobutamine-induced decrease in regional myocardial perfusion in nonocclusive coronary stenosis. J Am Coll Cardiol 2004;44:1664–71.
14. Armstrong G, Pasquet A, Fukamachi K, Cardon L, Olstad B, Marwick T. Use of peak systolic strain as an index of regional left ventricular function: comparison with tissue Doppler velocity during dobutamine stress and myocardial ischemia. J Am Soc Echocardiogr 2000;13:731–7.
15. Voigt JU, Lindenmeier G, Exner B, et al. Incidence and characteristics of segmental postsystolic longitudinal shortening in normal, acutely ischemic, and scarred myocardium. J Am Soc Echocardiogr 2003;16:415–23.
16. Langeland S, D'Hooge J, Claessens T, et al. RF-based two-dimensional cardiac strain estimation: a validation study in a tissue-mimicking phantom. IEEE Trans Ultrason Ferroelectr Freq Control 2004;51:1537–46.
17. Langeland S, D'Hooge J, Wouters PF, et al. Experimental validation of a new ultrasound method for the simultaneous assessment of radial and longitudinal myocardial deformation independent of insonation angle. Circulation 2005;112:2157–62.
18. Greenbaum RA, Ho SY, Gibson DG, Becker AE, Anderson RH. Left ventricular fibre architecture in man. Br. Heart J 1981;45:248–63.
19. Liang H-Y, Cauduro S, Pellikka P, et al. Usefulness of two-dimensional speckle strain for evaluation of left ventricular diastolic deformation in patients with coronary artery disease. Am J Cardiol 2006:98:1581–6.