Echocardiographic strain imaging was introduced by Heimdal et al in 1998 as a means to calculate myocardial regional function from tissue Doppler velocity data.\(^2^,3\) Strain imaging is a variation of the previous concept of tissue Doppler myocardial velocity gradient, which calculates regional thickening independent of passive whole heart motion.\(^4^,5\) Because routine clinical evaluation of regional function is usually by visual wall-motion assessment, strain imaging has great promise to improve objective quantification of regional function. Strain imaging calculates deformation, which translates clinically to percent shortening in the longitudinal dimension (assessed from apical views) or percent thickening in the radial dimension (assessed from parasternal views).\(^1^,6^,7\) Theoretically, it has the important advantage of differentiating active myocardial motion from passive translational or tethering movements that other echocardiographic-Doppler approaches cannot differentiate. Indeed, there have been literally hundreds of publications over the last 10 years detailing a wide range of experimental and potential clinical applications of strain imaging; however, currently, few centers have adopted strain imaging in their routine clinical practice, preferring visual “eyeball” assessment, even though this is inherently subjective and requires a high level of experience and training.\(^8\) An important question remains: Is it now time for strain imaging to be adopted in a widespread clinical sense?

Two important articles are contained in this issue of *Circulation* that address the application of strain imaging as a tool to assess myocardial viability: an animal study of experimental ischemia and infarction by Lyseggen et al\(^9\) and a clinical study using dobutamine echocardiography with outcome data after revascularization by Hanekom et al\(^10\). The article by Lyseggen et al illustrates the additive value of strain imaging in an elegant open-chest animal model of coronary occlusion and reperfusion. Echocardiographic strain imaging was performed along with simultaneous dimensional measurements by myocardial crystals, which may be regarded as the “gold standard” of reference for regional function, during left anterior descending coronary occlusion of variable duration to induce differing degrees of transmural necrosis. Myocardial perfusion was quantified by radiolabeled microspheres, and strain imaging was repeated after reperfusion, followed by histological confirmation of the degree of necrosis and viable myocardium, respectively. Partially active myocardium was differentiated from passive increases in myocardial length with pressure-dimensional loops. The investigators found that the ratio of systolic lengthening to combined late and postsystolic shortening identified viable myocardium, and decreases in myocardial compliance (systolic lengthening/systolic pressure rise) defined necrotic myocardium. Strain imaging data were comparable to the invasively implanted myocardial crystal data. This study extends the work of Smiseth’s group, which has investigated strain imaging extensively, including its use to differentiate postsystolic thickening in ischemic disease as an active or passive phenomenon.\(^11^,12\) These studies have demonstrated that strain imaging may yield important physiological data; however, these and other animal studies used strain imaging under the best of conditions, with transducer placement for optimal signal-to-noise ratio and Doppler angle of incidence for longitudinal shortening and lengthening, which may not always directly translate into patient care scenarios.

Myocardial viability can be determined in patients with a dobutamine echocardiography protocol when either an improvement in resting wall motion or a biphasic response (initial improvement followed by worsening wall motion at high dose) was observed. This approach using visual wall-motion assessment has yielded sensitivities of 74% to 87% and specificities of 73% to 86% from experienced laboratories.\(^13^–^16\) The dobutamine echocardiographic viability study has gained clinical acceptance as an alternative to widely used radionuclide methods or emerging MRI approaches. However, difficulties remain in the subjectivity of echocardiographic visual wall-motion interpretation, particularly in patients with previous myocardial infarction, in whom passive tethering motion is a confounding variable. The study by Hanekom et al\(^10\) applied strain and strain rate imaging as an adjunct to visual assessment in a series of 55 patients who underwent a dobutamine echocardiography viability study and subsequently underwent percutaneous or surgical revascularization. They applied strain and strain rate imaging to the standard apical 4-chamber, apical 2-chamber, and apical long-axis views and combined these measures with routine visual wall-motion scoring.\(^17^,18\) The investigators then reassessed patients 9 months after percutaneous revascularization or coronary bypass surgery and defined a positive viability response as improvements in wall motion ≥4 segments or an overall increase ≥5% in ejection fraction units. Of the 42% of patients with improvements in ventricular function after
revascularization, strain and strain rate imaging combined with routine visual assessment increased sensitivity from 73% to 83% compared with visual assessment alone. Although specificity was unaffected, the study provided important clinical outcome data to support the additional yield of strain imaging to determine myocardial viability.

What are some of the obstacles that currently may be limiting the adoption of tissue Doppler strain imaging for routine clinical practice? Four principal limitations include the following: (1) Myofiber orientation is complex. Myofibers are arranged in a spiral configuration that involves longitudinal shortening, radial thickening, and rotational vectors, which must be examined in isolation using tomographic imaging techniques. Because strain occurs in 3 dimensions in reality, 1-dimensional strain may represent an oversimplification. (2) Tissue Doppler strain can only be measured along the path of the ultrasound beam. Because tissue Doppler strain is derived from relative changes in velocity along a single ultrasound scan line, it can only accurately assess shortening or thickening when this principal vector is aligned with the ultrasound beam. This is an important limitation in assessing left ventricular shortening in patients who have abnormal geometry, particularly spherical shapes, in which the vector of longitudinal shortening is varying along its curvature whereas the transducer is stationary with a fixed ultrasound beam angle. Alternate vectors moving into or out of the imaging plane are not realized. (3) Tissue Doppler strain is angle-dependent. Determination of strain from tissue Doppler data is impossible near the left ventricular apex from apical views and near 10 and 2 o’clock in the parasternal short-axis view, in which the Doppler angle of incidence is 90°, even with angle-corrected tissue Doppler technology. Furthermore, the signal-to-noise ratio deteriorates greatly when the angle approaches 90°. (4) High-quality imaging is necessary. Although the tissue Doppler velocity signals are robust, any potential errors above become apparent when strain or strain rate imaging is used with below-average image quality. Strain imaging may help to decrease noise by integrating tissue Doppler data over the cardiac cycle, but noise is amplified by strain rate imaging, which is expressed as the first derivative of velocity data. Despite these limitations, strain imaging has the advantage of having operator-independent data acquisition, and each successive refinement in signal processing and software programming has resulted in improvements in strain-imaging data quality and analysis.

It is logical that the strengths of strain imaging may surpass these physical limitations in specific clinical scenarios in which differentiation of passive motion from active myocardial shortening or thickening is critically important but not easily discernible by visual assessment. The determination of myocardial viability in response to dobutamine inotropic stimulation represents one such scenario, in which visual assessment is challenging because of tethering from adjacent segments compounded by a low amplitude of wall excursion and varying degrees of regional dysfunction in patients with ischemic disease. The use of strain and strain rate imaging combined with routine visual assessment increased sensitivity compared with the use of visual assessment alone. This supports the incremental value of strain imaging in this particular scenario. Another practical clinical scenario in which strain imaging adds incremental value is in the assessment of radial dyssynchrony in patients evaluated for cardiac resynchronization therapy. Dyssynchrony analysis of the timing of regional contraction is usually limited to similar types of patients with depressed ejection fraction, low-amplitude wall excursion, and varying degrees of passive tethering motion, particularly in the septum. Although M-mode echocardiography can quantify the timing of septal and posterior wall motion in dysynchronous patients and predict response to resynchronization therapy, it cannot differentiate active from passive wall motion, which may be complex. Radial strain imaging can add incremental value by helping identify the timing of active thickening from passive motion in these patients. This has been shown to be most useful when the Doppler angle of incidence is parallel to the vector of wall thickening, as seen in anteroseptal and posterior wall segments from the parasternal short-axis view.

Is it time for routine application of strain imaging in clinical practice? These studies using strain imaging to enhance detection of myocardial viability take it 1 step closer to becoming a reality. Assessment of regional function by echocardiography remains one of the most important technical challenges for the future, particularly in patients with prior myocardial infarction. These separate but related works by Lyseggan et al and Hanekom et al demonstrate the continual improvements in quantitative echocardiographic technology, specifically, the utility of strain imaging to assess myocardial viability, and offer the great promise of a favorable impact on the care of patients with ischemic heart disease.

Disclosures

None.

References

7. Jamal F, Strotmann J, Weidemann F, Kukulski T, D’hooge J, Bijnsens B, Van de Werf F, De Scheerder I, Sutherland GR. Noninvasive quantifi-


Key Words: Editorials • echocardiography • infarction • ischemia
Echocardiographic Strain Imaging for Myocardial Viability: An Improvement Over Visual Assessment?
John Gorcsan III

Circulation. 2005;112:3820-3822
doi: 10.1161/CIRCULATIONAHA.105.593467

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/112/25/3820

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/