

EDITORIAL COMMENT

# Longitudinal Strain

## “Think Globally, Track Locally”\*

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For decades, echocardiography has proved to be a well-established noninvasive imaging modality for bedside assessment of global left ventricular (LV) function in patients with heart failure (HF). The need for objective quantification of systolic LV pump function for prognostic purposes has led to the development of numerous echocardiographic indexes. LV ejection fraction (EF) is the most studied and clinically applied index of LV systolic function. Measurement of LVEF is currently the mainstay for many clinical and therapeutic decisions, while it is also widely used to decide on patient inclusion in large clinical trials. Despite the introduction of 3-dimensional echocardiography allowing more direct LV volume measurement than conventional 2-dimensional (2D) methods, poor reproducibility of echocardiographic EF measurement remains a critical issue. Furthermore, it is well recognized that the prognostic value of volumetric LV systolic function assessment is limited in patients with HF and preserved LVEF. The introduction of speckle tracking echocardiography (STE) enabled direct measurement of myocardial tissue deformation from conventional 2D B-mode images. Several studies have shown the usefulness of STE-derived global longitudinal strain (GLS) as a replacement of or an addition to LVEF for the prediction of outcome in different subgroups of HF patients (1-3).

In this issue of *JACC*, the paper by Sengeløv et al. (4) strengthens the evidence that GLS is a strong predictor of outcome in HF patients. In this first large cohort study of HF patients with reduced LVEF ( $\leq 45\%$ ) selected using wide inclusion criteria, they reported GLS to be the only echocardiographic parameter that remained independently predictive of all-cause mortality after adjustment for potential covariates. However, although the amount of statistical evidence of the predictive power of GLS and its superiority over conventional echocardiographic function parameters is growing, our current physiological understanding of the similarities and the differences between GLS and other parameters such as LVEF is limited.

SEE PAGE 1351

GLS is 1 of the 3 principle strains of the LV, the other 2 being global circumferential (GCS) and global radial strain (GRS). These principal strains are kinematically coupled to changes in LV cavity volume. Whereas EF is purely based on measurement of end-diastolic and end-systolic LV cavity volumes (EDV and ESV, respectively), the relationship between the principal strains and LV cavity volume is influenced by the size of the LV wall. This can be more easily appreciated while focusing on LV fiber strain ( $\epsilon_f$ ), being the strain in the fiber direction. As proposed by Arts et al. (5) and validated by Delhaas et al. (6), change in LV fiber strain ( $\Delta\epsilon_f$ ) during ejection relates to both LV cavity volume and left ventricular wall volume (LVWV) as follows:

$$\Delta\epsilon_f = \frac{1}{3} \ln \left( \frac{1 + 3\text{ESV}/\text{LVWV}}{1 + 3\text{EDV}/\text{LVWV}} \right)$$

Consider 3 different hearts (Figure 1) with a stroke volume of 70 ml, i.e., a normal healthy heart, a heart with concentric hypertrophy, and 1 with dilated cardiomyopathy. Although the LVEF is the same for the normal and the concentric hypertrophy cases, and LVWV does not differ between the dilated

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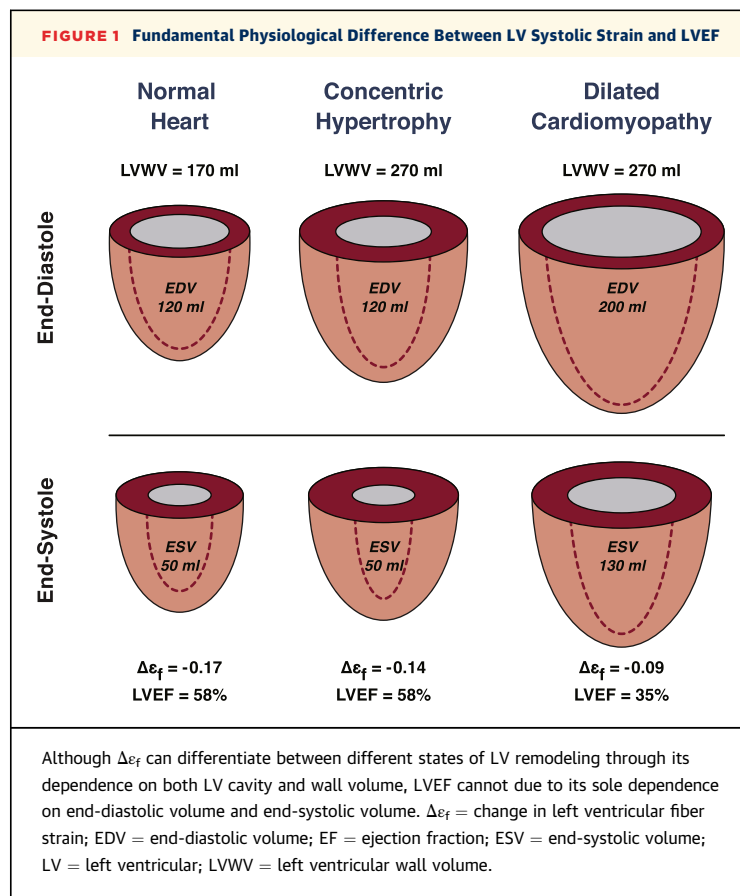
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cardiomyopathy and the concentric hypertrophy cases,  $\Delta\epsilon_f$  is different for all 3 cases (Figure 1). This dependence of systolic fiber strain, and hence GLS, on the ratio of LV cavity to wall volume may explain why GLS is independently associated with outcome in HF patients with preserved LVEF (1,3) as well as in those with reduced LVEF (2,4).

Knowledge of the myocardial tissue architecture is essential for understanding the relative contributions of different myocardial layers to the longitudinal, circumferential, radial, and torsional components of LV myocardial deformation. Myofiber orientation around the LV cavity changes gradually across the wall from a right-handed helical path in the sub-endocardium to a circumferential orientation in the midwall and a left-handed helical path in the sub-epicardium. Given this transmural change in myofiber orientation, it is plausible that GLS predominantly reflects the contractile function of the subepicardial and subendocardial layers of the LV wall. As the subendocardium is particularly susceptible to ischemia, stunning (7), or mechanical overload due to aortic stenosis or aging (8,9), GLS is likely to decrease with local or global loss of subendocardial contractile function in earlier stages of disease.

Using a CART (Classification And Regression Tree) analysis, Sengeløv et al. (4) propose that GLS can be used for risk stratification together with conventional echocardiographic function measurements. However, using a cutoff value for patient-specific risk assessment is only reliable if measurement of GLS is sufficiently standardized. The Sengeløv et al. (4) study used one and the same ultrasound system, but a significant limitation of the current implementation of 2D STE is the difference in strain values calculated by the different vendors (10,11). Importantly, a joint effort between the American Society of Echocardiography, European Association of Cardiovascular Imaging, and industrial partners currently addresses the issue of intervendor variability in STE strain measurement. In a consensus document (12), this Task Force identified potential sources of measurement variability and formulated technical recommendations to improve reproducibility of STE-based deformation imaging. In addition, practical recommendations aiming at a common standard for GLS quantification have recently been published in this journal (13).

In the search for a new diagnostic LV systolic function index representing the overall tissue's contractility, one always has to investigate its dependence on hemodynamic boundary conditions such as preload and afterload. Several studies have shown that LVEF is dependent on both preload and afterload (14,15). Few data are available on GLS and its dependence on



momentary loading conditions, although one may expect a strong dependence on preload in hearts with a normal ventricular myofilament length-dependent activation (16). Also longitudinal LV function has been shown to depend on afterload (17). To the best of our knowledge, no comprehensive study exists on the sensitivities of both LVEF and GLS to changes in preload, afterload, and contractility. Although pure changes of preload, afterload, or contractility are hard to establish in vivo, computer models can be useful because they enable fast simulation of LV mechanics and hemodynamics under strict control of hemodynamic loading conditions. Here, we used the CircAdapt model of the closed-loop cardiovascular system (18,19) to simulate changes of preload (venous return) and afterload (mean arterial pressure) in a normal heart and a failing heart. The simulation of the normal heart was obtained as described previously (18). The failing heart was simulated by reducing ventricular contractility, i.e., the innate ability of the myofibers to generate tensile force, to 50% of its normal value (20). Starting from both baseline simulations (heart rate of 70 beats/min, mean arterial pressure of 100 mm Hg, and venous return of 5 l/min), preload was changed by

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