Circulation

AHA SCIENTIFIC STATEMENT

Speckle-Tracking Strain Echocardiography for the Assessment of Left Ventricular Structure and Function: A Scientific Statement From the American Heart Association

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ABSTRACT: Assessment of left ventricular systolic function is essential for diagnosing and managing cardiac diseases and provides important prognostic information to the treating clinician. However, traditional methods for assessing left ventricular systolic function such as ejection fraction are limited by their reliance on geometric assumptions, subjective reader interpretation, sensitivity to loading conditions and volume, and reflection of a single plane of motion. In addition to interobserver and intraobserver variability and technical confounders, this evaluation is complicated by the complex 3-dimensional organization of the myocardial fibers, which are oriented longitudinally in the subendocardium, transversely in the midmyocardium, and obliquely in the subepicardium. Conversely, 2-dimensional speckle-tracking echocardiography measures left ventricular deformation as myocardial strain in the 3 planes of chamber motion: longitudinal, circumferential, and radial. From a clinical perspective, left ventricular global longitudinal strain offers superior diagnostic and prognostic value across the spectrum of cardiovascular disorders compared with ejection fraction, is highly reproducible, and detects subclinical dysfunction before the ejection fraction declines. Given the expanding clinical utility of speckle-tracking echocardiography and the incremental prognostic and therapeutic value of integrating global longitudinal strain into clinical practice as a potential biomarker, the objectives of this scientific statement are (1) to review the principles and technical aspects of speckle-tracking echocardiography strain imaging; (2) to provide a practical, evidence-based review of the application of speckle-tracking echocardiography in heart failure, cardiomyopathies, ischemic heart disease, valvular disease, and cardio-oncology; (3) to explore the potential utility of speckle-tracking echocardiography in cardiac resynchronization and implantable cardioverter defibrillator therapy; and (4) to outline the future directions of speckle-tracking echocardiography.

Key Words: AHA Scientific Statements ■ cardio-oncology ■ cardiomyopathies ■ coronary artery disease ■ echocardiography ■ global longitudinal strain ■ heart valve diseases ■ hypertrophic cardiomyopathy

Assessing left ventricular (LV) systolic function is essential for diagnosing and managing cardiac diseases. The complex 3-dimensional organization of the myocardial fibers of the heart, which are arranged in a helical and perpendicular orientation, enables efficient "wringing-like" ejection, complicating traditional assessment. Fiber orientation varies throughout the myocardial wall, being longitudinal in the subendocardium, transverse in the midmyocardium, and oblique in the subepicardium.[1] Traditional methods for assessing LV systolic function such as LV ejection fraction (LVEF) are limited by their reliance on LV geometric assumptions, subjective reader interpretation, sensitivity to loading conditions and volume, and the fact that they primarily reflect a single plane of LV motion.[1]

In contrast, 2-dimensional speckle-tracking echocardiography (STE) measures myocardial strain, which assesses myocardial deformation during contraction and relaxation in the aforementioned longitudinal, circumferential, and radial planes and is expressed as a

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percentage change in myocardial length during the cardiac cycle. More specifically, LV global longitudinal strain (GLS) offers superior diagnostic and prognostic value across the spectrum of cardiovascular disorders compared with LVEF and is used in clinical practice for its sensitivity in detecting subclinical LV dysfunction often before LVEF declines.[2] The strengths of GLS by STE include its angle independence, sampling of all LV wall segments in a given view, high feasibility and reproducibility, and excellent spatial resolution.[1,3]

Joint statements were issued to standardize strain imaging with STE, aiming to reduce variability and to improve its clinical application.[3,4] In addition, recommendations for cardiac chamber quantification outlined best practices for measurement and reporting of strain.[5] Given the expanding clinical utility of STE and the incremental prognostic and therapeutic value of integrating GLS into clinical practice as a potential biomarker, the objectives of this scientific statement are (1) to review the principles and technical aspects of STE strain imaging; (2) to provide a practical, evidence-based review of the application of STE in heart failure (HF), cardiomyopathies, ischemic heart disease, valvular disease, and cardio-oncology; (3) to explore the potential utility of STE in cardiac resynchronization and implantable cardioverter defibrillator therapy; and (4) to outline the future directions of STE.

PRINCIPLES AND TECHNICAL CONSIDERATIONS

Overview of LV Anatomy, Deformation, and Mechanics

LV deformation and mechanics can be assessed in 3 planes of motion: longitudinal (shortening and lengthening), circumferential (shortening and lengthening), and radial (thickening and thinning) strain. As the subendocardial, midmyocardial, and subepicardial layers contract, the LV shortens and twists around its long axis to transmurally disperse shearing forces and to eject a systolic stroke volume. The subendocardial and subepicardial fibers are arranged in a helical orientation and obliquely to one another at a 60° angle, whereas the midmyocardium is arranged in an equatorial plane. It is important to note that the myocardial layers are bound by interstitium, and dysfunctional mechanics in 1 layer will affect the transmural mechanics to varying degrees.[6]

The subendocardial LV fibers are characterized by longitudinal motion, the midmyocardium by circumferential motion, and the subepicardium by longitudinal and circumferential torsional deformation. The LV maintains a normal ejection fraction in many pathological conditions, given that this measure of systolic function is strongly affected by the interplay between circumferential and radial LV mechanics, geometry, wall thickness, and loading conditions.[7] Conversely, impaired longitudinal deformation of the LV has been well validated as an early marker of subclinical LV impairment that occurs before overt systolic dysfunction (LVEF <50%). Clinically, LV GLS provides superior disease-specific diagnostic, prognostic, and treatment insights compared with LVEF and is intimately related to subendocardial dysfunction.[2] Last, although LVEF remains of prognostic value in various cardiovascular diseases, GLS strengthens this risk stratification and offers superior reliability for serial LV function assessment, with lower intraobserver and interobserver variability. This consistency holds even across physicians with varying expertise, making GLS a more dependable tool for serial monitoring of cardiac performance.[8]

A large patient-level meta-analysis has proposed a GLS >−16% to be the absolute threshold indicating myocardial dysfunction regardless of vendor or clinical covariates and should alert the clinician to carefully assess for cardiac pathology.[9] However, interpreting the assessment within the clinical context is paramount and of particular importance within the "gray zone" GLS values of −16% to −18%, which are considered borderline or low normal. Age, sex, loading conditions, and obesity are the most prevalent clinical modifiers to GLS that must be accounted for. Although the American Society of Echocardiography/European Association of Cardiovascular Imaging Strain Standardization Task Force has actively worked toward harmonizing GLS measurements across vendors and software platforms, full standardization has not yet been achieved. GLS interpretation should thus remain context dependent and incorporate patient demographics, vendor-specific differences, and longitudinal trends.

Basic Principles

Contemporary LV strain assessment is most commonly performed with 2-dimensional STE, whereby software algorithms track stable kernels of myocardial speckles (persistent artifacts) throughout the cardiac cycle. Strain represents the maximal deformation of the tracked LV segment normalized to its original length. Correct patient positioning is paramount for obtaining the optimal imaging windows for assessment, with frame rates of 40 to 90 frames per second providing appropriate resolution.[3,4] Clear visualization of the endocardial borders is necessary to ensure accurate tracking and estimation of GLS, which is performed and averaged from the apical 4, 2, and 3-chamber (apical long-axis) views.

Although the layered helical myocardial architecture prompts consideration of multilayer strain relevance, full (midwall) GLS currently predominates in clinical echocardiography. Transmural strain differences between the subendocardial and subepicardial layers are smaller for longitudinal strain compared with radial and circumferential strains. In addition, the myocardial layers are

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mechanically tethered, and echocardiographic lateral resolution is insufficient to differentiate layer-specific longitudinal strain reliably, with no consistent differential findings between normal and infarcted segments across multiple vendors.[10] Thus, there is insufficient evidence to recommend layer-specific LV strain for routine clinical use, and full midwall GLS remains the most preferred approach.[5]

In the selection of the region of interest for strain assessment, it is prudent to avoid apical foreshortening (overestimation of GLS due to geometric distortion and the apparent hypercontractility of the false apex) and tracking of the pericardium (underestimation of GLS due to tethering of the subepicardium). Abnormalities in LV chamber geometry and wall thickness such as interventricular septal bulging and asymmetric thickness may influence strain measurements. The region of interest to assess GLS should be set straight and longitudinally, excluding focal septal bulging. However, in ventricles with asymmetric thickening in >1 continuous wall segment, care should be taken to widen the region of interest for inclusion. If specific segments display poor tracking, it is critical to reimage or adjust the region of interest manually.[9]

Common Pitfalls and Solutions

A typical assessment output will include a GLS polar map, region of interest, segmental strain values and curves, and M-mode strain depiction when applicable (Figure 1). Although it is important to acknowledge that a component of intervendor variability in GLS measurement exists, a dedicated task force comprising cardiovascular imaging experts and industry representatives has made significant progress in standardizing the software, reporting, and interpretation of cardiac deformation imaging.[3–5] One important caveat when interpreting GLS across vendors is whether tracking was applied to the LV endocardium only compared with full-wall thickness.[4,7,9] Longitudinal myocardial fibers predominate in the subendocardium, and with the additive effect of the ellipsoid LV geometry, tracking only in this layer overestimates GLS. As previously mentioned, the 3 myocardial layers are bound through interstitial networks, and their mechanics are mutually inclusive. Thus, it is prudent to document whether GLS is assessed through endocardium only or full-wall tracking and to use caution when comparing values across vendors with differing myocardial tracking algorithms.

Cardiac event timing is required and, depending on the vendor, may be performed with LV outflow tract Doppler, manual or automatic selection of the aortic valve closure point, or triggering of systolic image acquisition by gating to the R wave of the ECG. The last option appears to be the most frequently used, and care must be taken because if the R wave is not detected accurately, strain measurements could be mistimed and inaccurate. In addition, a paced rhythm with a prominent atrial pacer spike can be mistaken for a QRS complex and incorrectly time the strain measurements, resulting in inaccurate assessment of myocardial deformation. A limitation of 2-dimensional STE is the need for consistent R-R intervals, restricting its use in arrhythmias or respiratory variations. Real-time triplane

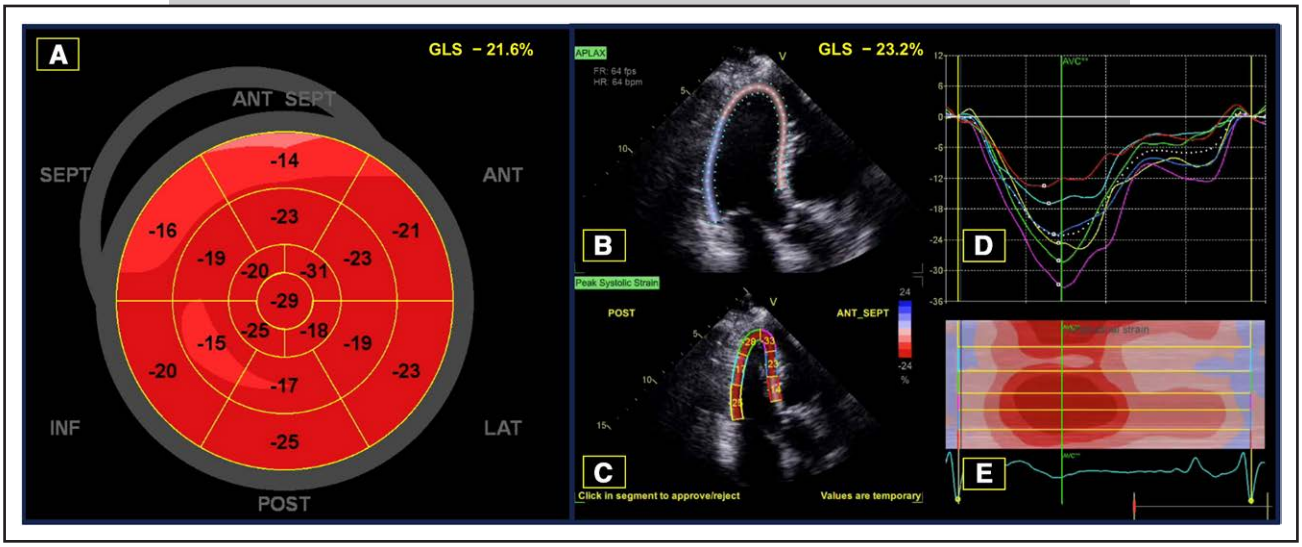

Figure 1. Example of a longitudinal strain assessment output in a patient with normal physiology.
A, Polar longitudinal strain map depicting the peak systolic strain in each of the 17 myocardial segments, as well as the global longitudinal strain of −21.6%. **B** and **C**, Region of interest and segmental color-coded peak systolic stain values of the apical 3-chamber left ventricular myocardial segments. **D**, Color-coded strain curves of each apical 3-chamber myocardial wall segment. **E**, M-mode representation of the longitudinal strain. ANT indicates anterior; ANT SEPT, anteroseptal; APLAX, apical long axis; AVC, aortic valve closure; GLS, global longitudinal strain; INF, inferior; LAT, lateral; POST, posterior; and SEPT, septal.

echocardiography with a matrix probe overcomes this by acquiring 3 simultaneous LV views from a single cycle. Last, to reduce motion artifacts and to improve image quality, the patient is asked to inhale or exhale and hold their breath while the images are acquired. Capturing 3 consecutive beats ensures that the data are reliable and reproducible.

Documentation of the systemic blood pressure at the time of strain assessment is important because GLS can be attenuated by increased afterload, particularly in patients with hypertension, aortic stenosis (AS), and hypertrophic cardiomyopathy.[9] Indeed, experimental models using graded aortic banding have shown a moderate relationship between increasing LV wall stress and worsening GLS (r=0.68, P<0.005) and radial strain (r=0.5, P=0.02), although to a lesser extent than impacts classically observed on LVEF.[11] Akin to LV pressure-volume loops, integrating the systemic blood pressure as measured by the brachial artery cuff pressure with GLS produces a stress-strain loop that estimates the global and regional LV myocardial work performed. This novel echocardiographic parameter was designed to resolve whether GLS reduction is due to reduced contractility (reflected as reduced myocardial work) or increased afterload (reflected as increased myocardial work).

For accurate LV myocardial work analysis, the brachial artery cuff pressure is measured immediately after recording of the apical views, with the patient supine and relaxed with the arm at the level of the heart, to avoid overestimation from prestudy blood pressure (eg, emotional stress) or underestimation in the left lateral positioning (eg, arm height).[12] Myocardial work parameters have been shown to discriminate coronary ischemia and to predict a positive response to cardiac resynchronization therapy (CRT).[13–15] From a physiological standpoint, myocardial work indices reflect cellular glucose metabolism, oxygen consumption, and tissue fibrosis, offering a promising adjunct modality to LV performance assessment as clinical experience and intervendor development progress.[16]

HF AND CARDIOMYOPATHIES

HF is a complex syndrome characterized by structural or functional cardiac abnormality and corroborated by elevated natriuretic peptide levels or objective evidence of pulmonary or systemic congestion.[17] Echocardiography remains the standard diagnostic tool for categorizing HF according to LVEF. However, LVEF has important limitations such as observer variability and geometric assumptions, particularly in HF with preserved ejection fraction (HFpEF). In HF populations, GLS has emerged as an important metric for detecting subclinical myocardial dysfunction, differentiating pathological conditions, and monitoring therapeutic responses (Figure 2).

Preclinical HF

Preclinical HF, defined as stage B, offers a key opportunity for early intervention. According to the 2022 American Heart Association/American College of Cardiology guidelines, a GLS value ≥−16% in the setting of LVEF >50% is recommended for diagnosing stage B HF.[18] GLS provides prognostic value in preclinical HF by identifying subclinical myocardial dysfunction before the onset of overt symptoms and may reclassify the HF stage in up to 14% of patients.[19]

HF With Preserved Ejection Fraction

HFpEF, in which LVEF remains ≥50%, accounts for >50% of HF cases.[18] The European Society of Cardiology guidelines acknowledge GLS ≥−16% as a minor diagnostic criterion for HFpEF.[17] This is of paramount utility, given the overlap of HFpEF and noncardiac causes of dyspnea in terms of clinical presentation, which often delays diagnosis. Analyses from randomized controlled trials have correlated impaired GLS in HFpEF with increased LV stiffness, elevated NT-proBNP (N-terminal pro-B-type natriuretic peptide) levels, and greater adverse cardiovascular outcomes independently of established clinical risk factors.[20,21]

HF With Reduced Ejection Fraction

In HF with reduced ejection fraction, GLS offers predictive accuracy that exceeds LVEF in terms of mortality and hospitalizations.[22–24] In a study of 1065 patients with HF with reduced ejection fraction, each 1% absolute decrease in GLS conferred a 15% increased risk of all-cause mortality (P=0.008), with the worst outcomes among men with atrial fibrillation.[22] Worsening GLS correlates with LV remodeling, progression of diastolic dysfunction, and worsening functional class, with a cutoff of ≥−6.95% associated with >2-fold increased risk of adverse cardiovascular events regardless of an ischemic or nonischemic substrate (P=0.01).[23]

Hypertrophic Cardiomyopathy

Hypertrophic cardiomyopathy is a heterogeneous disorder characterized by LV hypertrophy, myocardial fiber disarray, and extensive fibrosis. In early or mild phenotypic stages, distinguishing hypertrophic cardiomyopathy from other causes of hypertrophy is challenging with important clinical implications. It is important to note that a GLS of ≥−14.3% can differentiate hypertrophic cardiomyopathy from hypertensive heart disease or athletic remodeling with a sensitivity, specificity, and predictive accuracy of 77%, 97%, and 87%, respectively (P<0.001).[25] GLS and myocardial work indices provide excellent risk stratification in hypertrophic cardiomyopathy, with a GLS ≥−10% increasing the occurrence of major adverse cardiovascular events 4-fold compared

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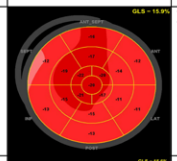
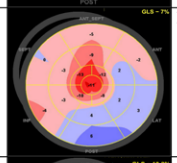
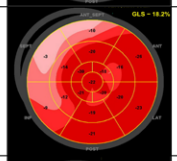
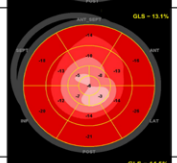
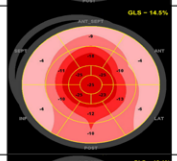
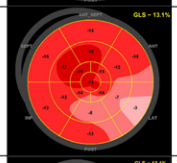
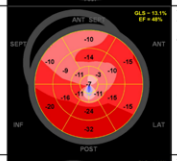
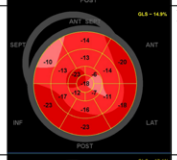
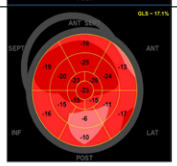
Disease	GLS Polar Map	Key GLS Association
Pre–clinical heart failure		Figure: GLS −15.9% consistent with subclinical LV dysfunction. • GLS > −16% reclassifies 14% from stage A to B heart failure. • Incremental prognostic value beyond LVEF. • Independently associated with heart failure hospitalization or death.
Heart failure with preserved ejection fraction		Figure: GLS −15.6% consistent with subclinical LV dysfunction. • GLS > −16% is a minor diagnostic criterion per ESC guidelines. • Correlates with LV stiffness and worsening heart failure markers independent of clinical covariates.
Heart failure with reduced ejection fraction		Figure: GLS −7% in a patient with LVEF 30% and advanced dilated cardiomyopathy. • 15% increased risk of mortality per 1% absolute GLS reduction • Worsening GLS correlates with LV remodeling, diastolic dysfunction, and heart failure symptoms • GLS > − 6.95% associated with > 2-fold increased risk of adverse events regardless of an ischemic or nonischemic cardiomyopathy
Hypertrophic cardiomyopathy (sigmoid septum morphology)		Figure: GLS −18.2% with marked focal impairment in the basal interventricular septum and inferior territory. • GLS > −14.3% differentiates HCM from hypertensive heart disease or athletic remodeling (sensitivity 77%, specificity 97%) • GLS > −10% associated with 4−fold increased risk of death • Polar map GLS patterns correlate with phenotypic expression
Hypertrophic cardiomyopathy (apical variant)		Figure: GLS −13.1% with marked impairment of mechanics in the apical segments. • HCM morphology with highest prevalence of impaired GLS • Global wasted work >186 mmHg% strongly predictive of adverse outcomes • Apical aneurysm associated with lower global work efficiency
Cardiac amyloidosis		Figure: GLS −14.5% with a relative apical sparing pattern (reduced strain in the basal and mid LV segments with preserved apical strain). • Relative apical sparing = apical/(basal + mid) longitudinal strain • Ratio >1 predicts cardiac amyloidosis (sensitivity 93%, specificity 82%) • Clinical context important: occurs in up to 44% of aortic stenosis patients
Anderson–Fabry disease		Figure: GLS −13.1% with greatest impairment in the basal to mid lateral and posterior LV territory • Polar GLS map pattern of basal to mid posterior−lateral impairment common • GLS > −14.1% predicts increased risk of adverse outcomes
Ischemic heart disease		Figure: GLS −13.1% affecting the septal, anterior, and lateral LV territories after a large anterior myocardial infarction • Pattern of GLS impairment correlates with involved coronary artery territory • In STEMI, GLS > −15% predicts LV dilatation and remodeling at 6 months • In non−STEMI, GLS > −16.5% identifies obstructive lesions and correlates with the number of diseased arteries
Cardio−Oncology		Figure: GLS −14.9% pre−anthracycline therapy indicating a higher risk patient • GLS > −15% before anthracycline correlates with HF risk and all cause mortality better than LVEF <50% • GLS is particularly useful with low normal LVEF between 50% to 59% • A relative 15% worsening in GLS during chemotherapy indicates subclinical LV dysfunction and may guide cardioprotective therapies
Valvular heart disease		Figure: GLS −17.1% in severe mitral regurgitation indicating a higher risk patient • Aortic stenosis: GLS > −15% is highly predictive of LV replacement fibrosis and all cause mortality • Aortic regurgitation: GLS > −19.5% mediates long term survival in asymptomatic severe insufficiency • Mitral regurgitation: GLS > − 21.7% predicts mortality in the setting of preserved LV geometry and LVEF

Figure 2. Summary and polar map example of left ventricular global longitudinal strain assessment.
ESC indicates European Society of Cardiology; GLS, global longitudinal strain; HCM, hypertrophic cardiomyopathy; HF, heart failure; LV, left ventricle; LVEF, left ventricular ejection fraction; and STEMI, ST-segment–elevation myocardial infarction.

with a GLS <−16% (P<0.001) and offer insight into the impact of structural changes such as apical aneurysms.[26–28] Last, GLS polar maps improve diagnostic accuracy and user confidence in that unique patterns identify specific pathologies and phenotypic expressions such as significant GLS impairment in the anteroseptum

and inferoseptum for reverse curve phenotype versus distal and apical LV impairment in the apical phenotype. In apical hypertrophic cardiomyopathy, the hypertrophied segments may mask the overt appearance of apical aneurysms, which can be recognized by their dyskinetic motion and "blueberry-on-top" GLS polar map pattern.[29]

Cardiac Amyloidosis

Infiltrative cardiomyopathy is characterized by the deposition of abnormal proteins, granulomas, and mineral elements, among other substances within the myocardium, resulting in progressive fibrosis and restrictive physiology. In cardiac amyloidosis, amyloid fibril deposition in the myocardium affects longitudinal deformation, resulting in dysfunctional mechanics.[30] A hallmark of cardiac amyloidosis is the "apical-sparing" strain pattern, characterized by reduced longitudinal strain in the basal and mid-LV segments with preservation at the apex. It is hypothesized to result from greater basal LV apoptosis and wall stress attributable to a larger regional chamber radius, greater interstitial space expansion at the basal LV territory, and a complex myocyte orientation at the apex. The relative apical sparing is quantified as apical/(basal+mid) longitudinal strain, and a ratio >1 has been shown to predict cardiac amyloidosis with a sensitivity and specificity of 93% and 82%, respectively (area under the curve, 0.94 [95% CI, 0.89–0.99]).[31] An apical longitudinal strain >−14.5% has also been proposed as a threshold for marked increase in major adverse cardiovascular events in a cohort of mixed amyloidosis subtypes.[30] Although most prevalent in cardiac amyloidosis (74%), a degree of apical sparing may occur in up to 44% of patients with AS, highlighting the importance of integrating ancillary echocardiographic findings and clinical context.[32] In addition, the presence of chronic kidney disease may reduce the specificity of GLS in detecting cardiac amyloidosis with the apical-sparing pattern.[33]

Anderson-Fabry Disease

Anderson-Fabry disease is a lysosomal storage disorder characterized by accumulation of intracellular glycosphingolipids, including infiltration within the myocardium. Patients with Anderson-Fabry disease often present with concentric LV and papillary muscle hypertrophy. Impaired GLS is associated with a higher incidence of major adverse cardiovascular events compared with preserved GLS, with a cutoff of −14.1% proposed as a robust threshold.[34] Polar maps with impaired GLS predominating in the basal to midlateral and posterior LV territory are often noted.[35]

ISCHEMIC HEART DISEASE

As noted here, LV mechanics are facilitated by the intricate helical structure of the myocardial fibers that support coordinated chamber contraction, relaxation, and systolic ejection, all of which rely on healthy transmural coronary blood supply. Subendocardial fibers are more vulnerable to myocardial ischemia, which can significantly impair cardiac function. In acute ischemic conditions, these fibers exhibit evidence of decreased systolic longitudinal shortening and postsystolic shortening after closure of the aortic valve.[36] The myocardial injury often manifests in the setting of preserved circumferential shortening and LVEF, resulting in regional LV deformational dysfunction (Figure 2).

Regional LV Deformation, Systolic Lengthening, and Postsystolic Shortening

Techniques such as STE-derived GLS allow a more nuanced evaluation of regional myocardial deformation. GLS is particularly effective in identifying regional abnormalities in myocardial function at rest and stress that may not be apparent through conventional imaging methods, and its reproducibility in patients with ischemic heart disease surpasses that of LVEF. A study of 47 patients with recent acute coronary syndrome compared the assessment of GLS and LVEF between expert and trainee echocardiographers and showed excellent correlation in GLS measures regardless of experience (intraclass correlation coefficient, 0.89; r=0.94) relative to LVEF (intraclass correlation coefficient, 0.74; r=0.71, P<0.0001).[37]

In the analysis of individual LV segmental strain curves, the observance of systolic lengthening informs the presence of ischemic myocardium, whereby affected myocardial segments elongate during systole as a result of ineffective contraction, dyskinetic motion caused by abnormal energetics, or transmural scarring. Similarly, postsystolic shortening occurs when LV segments contract after the primary systolic phase, often seen in ischemic myocardium (Figure 3A). In a prospective study of 293 patients with suspected angina, postsystolic shortening was an independent predictor of obstructive coronary artery disease and conferred a 2.5-fold increased risk of adverse cardiovascular events (P=0.03).[38] Given their utility, the 2024 European Society of Cardiology guidelines for the management of chronic coronary syndromes recommend strain imaging for detecting decreased systolic shortening or GLS, early systolic lengthening, or postsystolic shortening in patients with normal LV function and a clinical suspicion of coronary artery disease.[39]

Acute and Chronic Coronary Syndromes

Incorporating STE and GLS into the early assessment of acute coronary syndromes identifies patients who could benefit from early invasive management in that the aforementioned use of segmental strain curve analyses and interpretation of GLS polar maps can markedly

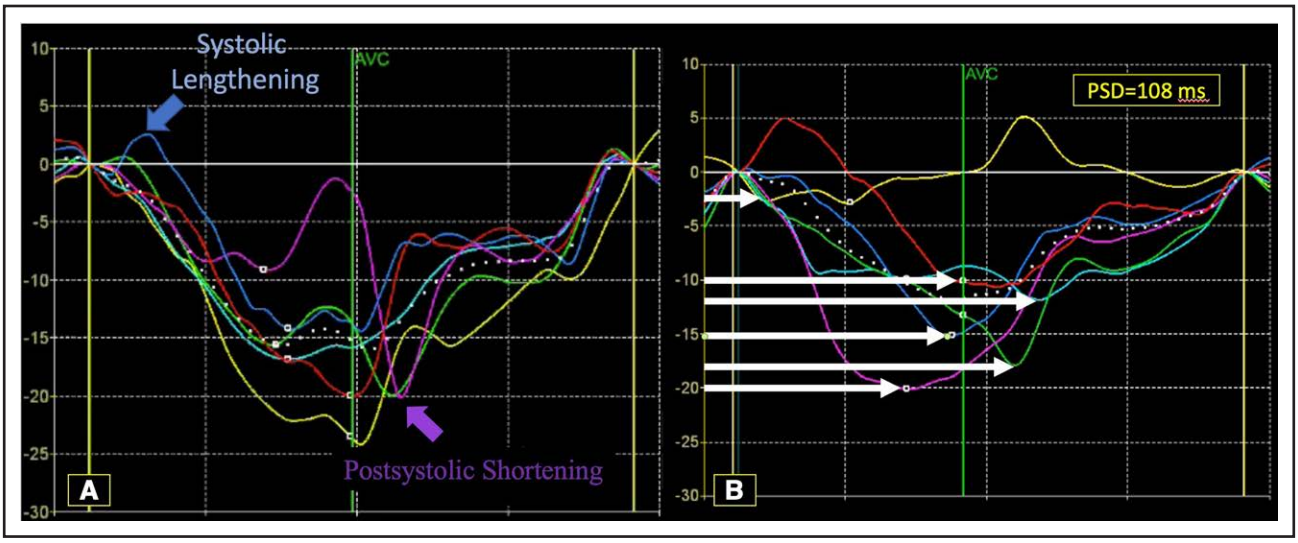


Figure 3. Utility of individual segmental longitudinal strain curve analysis.
A, A patient with ischemic heart disease exhibiting both systolic lengthening (blue curve with early positive longitudinal strain) and postsystolic shortening (purple curve with peak longitudinal stain occurring well after aortic valve closure and nearly double the value of systolic strain). **B**, A patient with significant left ventricular electromechanical dyssynchrony (variable peak longitudinal strain of the 6 myocardial segments of the specific view denoted by individual white arrows) and markedly increased mechanical dispersion (peak strain SD [PSD], 108 milliseconds). AVC indicates aortic valve closure.

enhance standard echocardiographic parameters used in evaluating for myocardial ischemia. GLS has been shown to be an important predictor of postdischarge adverse outcomes in patients with ST-segment–elevation myocardial infarction. In 1041 patients with ST-segment–elevation myocardial infarction with a mean LVEF of 47±9% treated with percutaneous coronary intervention, a GLS >−15% independently predicted LV dilatation and adverse remodeling at 6 months (P<0.001).[40] At a midterm follow-up of 5 years in 1060 patients with ischemic heart disease and prior myocardial infarction, a baseline GLS >−11.5% was predictive of poor survival, with each 5% relative change increasing risk by 1.6-fold (P<0.001).[41] In patients with non–ST-segment–elevation myocardial infarction, an impaired GLS of >−16.5% effectively identifies severe coronary obstruction, defined as obstructive lesions of >70%. The degree of GLS impairment directly correlates with the number of involved coronary arteries, enhancing risk stratification.[42]

CARDIO-ONCOLOGY

LV Functional Assessment and Cancer Therapy

Many anticancer agents are linked to cardiovascular side effects, most commonly LV dysfunction and HF, collectively called cancer therapeutics–related cardiac dysfunction (CTRCD). Given the impact of CTRCD on treatment and prognosis, current strategies focus on early detection and intervention to minimize myocardial injury during continued oncology care. Echocardiography is crucial in clinical decision-making before, during, and after cancer therapy (Figure 4).[43]

Surveillance of LVEF has been the main parameter for identifying LV dysfunction and defining cardiotoxicity. CTRCD is commonly defined as an absolute 5% LVEF reduction in symptomatic patients or a 10% decrease to <53% in asymptomatic patients. Early detection relies on serial echocardiography to detect LVEF decline before HF symptoms. However, LVEF has limitations, including measurement variability, and is a late indicator, often leading to suboptimal LV recovery even with cardioprotective therapy.[44] A large body of literature supports the diagnostic and predictive value of STE-assessed GLS in patients receiving potentially cardiotoxic therapy. As a result, guidelines, including the 2022 European Society of Cardiology guidelines on cardio-oncology, recommend incorporating GLS into the evaluation of patients throughout potentially cardiotoxic cancer therapy[45] (Figure 2).

Value of GLS Before, During, and After Cancer Therapy

Baseline GLS can identify high-risk patients and imply prognosis in those receiving anthracycline therapy. One study developed a baseline risk score model for predicting HF in patients with acute leukemia after anthracycline treatment, demonstrating that GLS >−15% correlated more strongly with HF risk than an LVEF <50%. In addition, a GLS >−15% was independently associated with all-cause mortality after adjustment for age and leukemia type (P<0.001), whereas cardiovascular disease and baseline LVEF <50% were not.[46] GLS is particularly useful for patients with LVEF in the lower

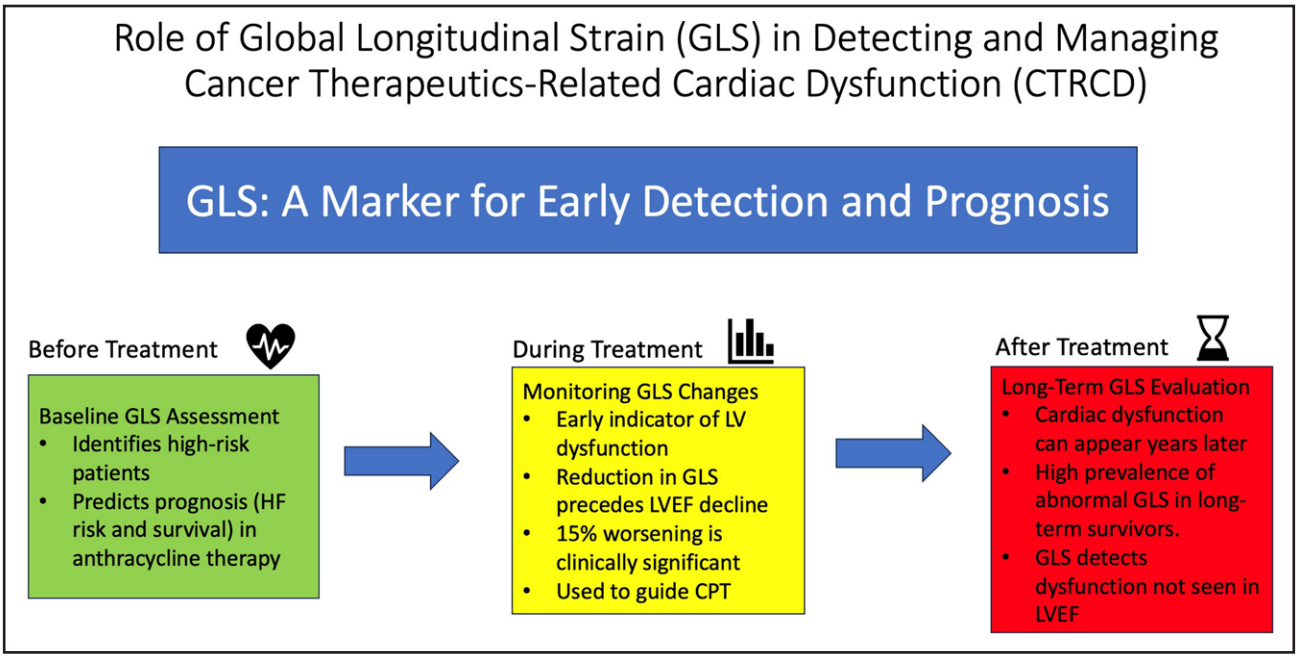


Figure 4. Utility of global longitudinal strain before, during, and after the use of cancer therapeutic agents.
CPT indicates cardioprotective therapy; HF, heart failure; LV, left ventricle; and LVEF, left ventricular ejection fraction.

limits of normal, between 50% and 59%, providing incremental prognostic value in predicting HF and overall mortality after anthracycline therapy.

Changes in GLS are well documented in patients receiving anthracyclines, anti–human epidermal growth factor receptor 2 therapy such as trastuzumab, or both. A meta-analysis of 21 studies involving 1782 patients with various cancer diagnoses found that both absolute and relative GLS decreases during treatment predict subsequent CTRCD. Notably, the identified cutoff values for GLS varied as a result of differences in sample sizes, CTRCD definitions, and potential publication bias. A relative 15% worsening in GLS is generally considered clinically significant and indicates subclinical LV dysfunction. These studies collectively confirm the prognostic value of GLS for cardiac dysfunction in patients treated with anthracyclines, trastuzumab, or both.[47]

Role of GLS in Guiding Cardioprotection and Late Follow-Up of Patients With Cancer

The role of GLS in guiding cardioprotective treatment during cardiotoxic therapy was explored in the SUCCOUR-MRI trial (Strain Surveillance during Chemotherapy for Improving Cardiovascular Outcomes) that included 355 patients, the majority of whom were women with breast cancer.[48] This randomized trial assessed the benefits of neurohormonal blockade in those with isolated GLS decline and no significant LVEF change during anthracycline treatment. Patients receiving cardioprotective medical therapy experienced less LVEF decline at 12 months (−2.5±5.4% versus −5.6±5.9%; P=0.009) and higher follow-up LVEF (59±5% versus 55±6%; P<0.0001) compared with patients receiving standard care. Thus, cardioprotective therapy for isolated GLS changes appears to be linked to less LVEF decline during anthracycline treatment in at-risk patients.

CTRCD can manifest years after treatment, particularly after anthracycline therapy or radiotherapy. The St. Jude Lifetime Cohort Study found that 31.8% of long-term adult survivors of childhood cancers treated with anthracyclines, chest radiation, or both exhibited cardiac dysfunction, indicated by decreased GLS, at a median of 23 years after diagnosis. Notably, 28% of survivors with normal LVEF showed cardiac dysfunction according to GLS, which was strongly linked to prior treatment exposure.[49]

VALVULAR HEART DISEASE

Valvular heart disease is characterized by progressive hemodynamic derangements in preload and afterload. LVEF is a key metric for determining the timing of intervention; however, its sensitivity to geometric factors and cardiac loading conditions makes interpretation challenging. Assessment of GLS in valvular heart disease allows the detection of subclinical LV dysfunction, particularly in patients with aortic and mitral regurgitation (MR) and AS, which supports timely intervention and improves patient outcomes (Figure 2).

Aortic Stenosis

In AS, LV pressure overload triggers compensatory hypertrophy to normalize wall stress, often resulting in

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preserved LVEF despite myocardial damage. Studies have demonstrated the utility of GLS in detecting subclinical LV dysfunction and associated clinical risk in AS. In a meta-analysis of 10 studies including 1067 asymptomatic patients with significant AS and LVEF ≥50%, impaired LV GLS was associated with a 2.5-fold increase in all-cause mortality risk, with an optimal cutoff value of >−14.7% ($P<0.0001$).[50]

With AS progression, the increased wall stress leads to diffuse interstitial collagen accumulation, evolving from reactive to replacement interstitial fibrosis. Both LV mass and myocardial fibrosis, detected with cardiac magnetic resonance (CMR) imaging, are independently associated with impaired GLS and portend adverse cardiovascular outcomes. In 261 patients with moderate to severe AS and preserved LVEF, a GLS >−15% predicted LV replacement fibrosis with 95% sensitivity independently of clinical risk factors, AS severity, and echocardiographic measures of LV mass and filling pressure ($P<0.001$).[51] Last, patients with moderate AS and preserved LVEF >50% but impaired GLS >−16% have been observed to have a 2-fold increased risk of mortality, which is similar to that of patients with an impaired LVEF <50%, highlighting the sensitivity of GLS assessment ($P<0.001$).[52]

Aortic Regurgitation

Chronic severe aortic regurgitation (AR) leads to LV volume overload, resulting in eccentric hypertrophy and LV dilatation to attenuate wall stress. Once the patient is symptomatic or LVEF declines, survival decreases significantly without timely surgical intervention. In a pivotal study, 84% of deaths in patients with severe AR occurred in patients with LV end-systolic diameter index <2 cm/m^2, indicating that risk increases even before overt LV remodeling.[53] In 1063 patients with asymptomatic severe AR, LV end-systolic diameter index <2.5 cm/m^2, and LVEF >50%, each unit of worsening GLS independently conferred an 11% increased risk of mortality ($P=0.003$). A GLS of >−19.5% was shown to mediate long-term survival, and aortic valve surgery at this threshold blunted mortality risk ($P<0.001$).[54]

Mitral Regurgitation

MR is characterized by pure volume overload, increased preload, and normal afterload, which allow LVEF to remain normal despite underlying dysfunction. In 737 patients with asymptomatic primary severe MR, preserved LVEF, and normal LV dimensions, a GLS >−21.7% was associated with increased mortality, suggesting that even "normal" GLS in severe MR is linked to poorer outcomes.[55] Each unit of worsening GLS independently conferred a 60% increased risk of long-term mortality ($P<0.001$), and addition of GLS to traditional risk models improved their predictive value. In dilated cardiomyopathy with severe secondary MR, GLS has been shown to reflect LV dysfunction more accurately than LVEF, although larger studies on its clinical utility are warranted.[56] Last, in 155 patients with symptomatic severe MR and preserved LVEF undergoing transcatheter edge-to-edge repair, a preprocedural GLS of >−14.5% conferred a higher 1-year mortality independently of MR cause and Society of Thoracic Surgeons risk score ($P<0.001$), providing incremental prognostic value and identifying patients at higher risk.[57]

CRT AND IMPLANTABLE CARDIOVERTER DEFIBRILLATOR THERAPY

In patients with cardiomyopathy meeting appropriate criteria, CRT and implantable cardioverter defibrillators are recommended by societal guidelines for the primary and secondary prevention of ventricular arrhythmias and sudden cardiac death and to improve electromechanical function of the LV in the setting of symptomatic HF and maximal guideline-directed medical therapy. The use of STE has proved invaluable in further understanding the complex pathophysiology across the spectrum of cardiomyopathic causes and provides useful measures for clinical prognostication and for predicting response to CRT and LV reverse remodeling.

When GLS is assessed by STE, the measure of LV mechanical dispersion is calculated as the SD of the time to peak longitudinal strain in each of the 17 myocardial wall segments analyzed (Figure 3B). Mechanical dispersion quantifies the temporal heterogeneity in electromechanical activation of the LV, serving as a surrogate for myocardial dyssynchrony and fibrosis. A mechanical dispersion >60 milliseconds is generally agreed on as the threshold for abnormal, identifies across cardiomyopathic substrates as a reproducible cutoff to predict ventricular arrhythmias, and is associated with mortality risk in severe AS.[58,59]

Radial strain STE describes the systolic LV wall thickening that occurs as a result of longitudinal and circumferential myofiber shortening and finite compressibility of the myocardium. Global radial strain is measured at the LV basal, midpapillary muscle, and apical levels and may be further analyzed regionally or segmentally. In patients with HF with reduced ejection fraction and LVEF <35% undergoing CRT device implantation, radial strain curves can quantify the degree of LV dyssynchrony and identify the latest activated myocardial segment, which may assist in selection of the optimal LV pacing site. The TARGET trial (Targeted Left Ventricular Lead Placement to Guide Cardiac Resynchronization Therapy) randomized 220 patients undergoing CRT implantation to LV lead placement guided by the STE radial strain delay of the latest activated myocardial segment free from scar or fluoroscopic lead placement.[60] STE guidance was associated with a greater

number of patients experiencing LV reverse remodeling (70% versus 55%; *P*=0.03), higher incidence of clinical improvement by ≥1 functional classes (83 versus 65%; *P*=0.003), and greater freedom from death or HF hospitalization (*P*=0.03) at the midterm follow-up. Similar findings were reported in the STARTER trial (Speckle Tracking Assisted Resynchronization Therapy for Electrode Region) of 187 patients randomized to radial STE-guided compared with fluoroscopic lead implantation, in which the benefit appeared magnified with a more narrowed QRS interval ≤159 milliseconds (*P*=0.004).[61] Thus, when a CRT device is implanted, an STE-guided approach using radial strain may allow more precise site selection for LV pacing to enhance reverse remodeling and to improve electromechanical synchrony. The measure of LV mechanical dispersion as part of the GLS assessment allows quantitative assessment of this latter point.

SUMMARY AND FUTURE DIRECTIONS

As the field of cardiac imaging rapidly evolves, it is important to identify future directions with regard to STE. It is important to note that the interest in and efforts to integrate LV strain assessment as a routine and standard practice across echocardiography laboratories are growing. By identifying early or subclinical LV dysfunction, strain imaging will allow individual treatment planning, optimization of timing for interventions, and nuanced risk stratification. Impaired LV GLS consistently shows an independent association with adverse outcomes, justifying its current use for risk stratification. However, outside of HF and CTRCD, evidence supporting the use of GLS to guide specific changes in clinical management is still evolving.

Machine-learning algorithms have shown that automated measurement of LV GLS may be performed within 8 seconds, which would support easy routine integration into laboratory workflow.[62] Artificial intelligence and deep learning enhance STE, with automated GLS and regional strain showing good agreement with conventional methods, accurately identifying subtle LV dysfunction, and offering rapid, vendor-agnostic results with less variability. Future validation and prognostic refinement of artificial intelligence–derived strain promise to elevate its clinical impact, making it a widely accessible tool for personalized care.[63–65] Nevertheless, it is acknowledged that challenges persist in educating nonimaging cardiologists and internal medicine physicians unfamiliar with the interpretation of strain imaging, necessitating structured training, workshops, and specialist-clinician collaboration to boost adoption.

CMR imaging techniques such as feature tracking and tagging offer alternative approaches to STE for the assessment of LV strain. There is particular value in CMR when assessing cardiomyopathies because of its relative independence from acoustic windows, larger fields of view, and ability to discern myocardial scarring and fibrosis. CMR feature tracking and tagging show good overall agreement with STE for GLS and circumferential strain, and CMR outperforms STE in detecting regional strain in infarcted myocardium.[66] However, CMR feature tracking has longer acquisition times, poorer spatial and temporal resolution, significant intervendor variability, and lower strain values compared with STE. It also relies on endocardial and epicardial contours rather than intramyocardial speckles, leading to high segmental strain variability that limits clinical use. The high cost and limited availability of CMR and patient factors such as claustrophobia and prohibitive device implantations also restrict its widespread application. Normal reference values for CMR strain imaging are essential before widespread clinical implementation.

The development of 3-dimensional echocardiography is being expanded to STE, which may enable a more accurate assessment of myocardial strain. Unlike 2-dimensional STE, which relies on multiple planes and may miss out-of-plane motion as a result of ventricular twisting, 3-dimensional STE captures all strain components—longitudinal, circumferential, and radial—in 1 acquisition, better reflecting the complex 3-dimensional organization of myocardial fibers, reducing acquisition time, and providing dynamic GLS polar maps while minimizing nonmyocardial speckle interference. However, the lower spatial and temporal resolution of 3-dimensional STE can limit tracking reliability, particularly for segmental analysis, and current limitations may underestimate 3-dimensional global strain compared with 2-dimensional values, posing ongoing challenges.[9]

Continued research and outreach by STE experts and collaborations between vendors have enabled significant advancements in identifying normative strain values across different populations, accounting for sex, age, and ethnicity, and have significantly improved intervendor comparability. As the interest in and clinical comfort with STE grow, so will its role in assessing the impact of the heart on cardiovascular (congenital heart disease, myocarditis, inflammatory disorders) and noncardiovascular (chronic kidney disease, sepsis, pulmonary disorders) conditions, which will further expand the use of this modality in everyday medical practice. An in-depth, multidisciplinary, stepwise recommendation of STE is needed to further the modality and to highlight its importance in the assessment of myocardial function.

ARTICLE INFORMATION

The American Heart Association makes every effort to avoid any actual or potential conflicts of interest that may arise as a result of an outside relationship or a personal, professional, or business interest of a member of the writing panel. Specifically, all members of the writing group are required to complete and submit a Disclosure Questionnaire showing all such relationships that might be perceived as real or potential conflicts of interest.

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*Modest.

†Significant.

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*Modest.

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