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Review

A Review of Alternative Measurements in Strain Imaging for Ventricular Arrhythmia Prediction

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Abstract

Global longitudinal strain has been established as a reliable tool to assess global left ventricular function and a marker of subclinical left ventricular dysfunction unrecognized by the ejection fraction. On the other hand, ventricular arrhythmias are the most common cause of sudden cardiac death. Their early detection is a challenge. Possible prognostic markers for the risk of ventricular arrhythmias are discussed in the literature – electrocardiographic, cardiac magnetic resonance, computed tomography, radionuclide imaging, and markers from new echocardiographic techniques. Of the latter, at this stage of knowledge, several markers have been discussed as informative for predicting ventricular arrhythmias – global longitudinal strain, radial strain and mechanical dispersion, and most recently, myocardial work. As far as we are informed, global longitudinal strain is particularly useful in patients with normal echocardiographic parameters such as left ventricular ejection fraction, left atrial diameter, left ventricular wall thickness, and aortic root. The relationship between mechanical dispersion and ventricular arrhythmias has been widely studied. The relationship has been studied more in some patient populations – heart failure, ischemic heart disease, long QT syndrome and arrhythmogenic cardiomyopathy, congenital heart disease. The role of mechanical dispersion as a predictor of ventricular arrhythmias in metabolic syndrome is scarce.

Keywords

ejection fraction, global longitudinal strain, mechanical dispersion, myocardial work, radial strain

INTRODUCTION

Cardiac arrests in the emergency rooms are usually caused by disturbances in the heart rhythm such as ventricular fibrillation or ventricular tachycardia. The underlying mechanisms are multifactorial in nature. Heterogenous ventricular activation is most often due to cardiac fibrosis, an aftermath of tissue damage.

Echocardiography plays an important role in the evaluation of the structural substrate for arrhythmogenesis. The benefits are that it is noninvasive, easily accessible, and portable. Most often this is done by measuring the left ventricular ejection fraction (LVEF). For more than 60 years, LVEF has been a primary tool for assessing systolic function. Its limitations associated with volume-only assessment have been documented over time due to technical constraints, such as poor repeatability, geometric variation, and load dependence.[1] Therefore, ejection fraction is an indirect measure of systolic function. Data in the literature shows that left ventricular ejection fraction is not reliable. This is confirmed by the fact that a majority of patients preserve LVEF in 30% to 40% of cases of sudden cardiac death in patients with heart failure. Clinical practice needs to have a more reliable parameter than LVEF to stratify the risk of ventricular arrhythmias.

Speckle-tracking echocardiography has several advantages for assessing myocardial function. It enables a thorough assessment of cardiac mechanics since strain measurement is extremely sensitive in detecting systolic dysfunction that the ejection fraction cannot detect.[2-4] Global longitudinal strain (GLS) has been established as a reliable tool for assessing global LV function and a marker of subclinical LV dysfunction over the past 15 years.[5]

On the other hand, early identification of patients at risk for ventricular arrhythmias is difficult in clinical practice, particularly in patients with LVEF >35%, despite the fact that ventricular arrhythmias are the most common cause of sudden cardiac death.[6-10] Possible prognostic markers for ventricular arrhythmia risk are discussed in the literature –electrocardiographic, cardiac magnetic resonance markers, computed tomography, radionuclide imaging and markers from new echocardiographic techniques.[8-20] Several of these markers are discussed as informative for predicting ventricular arrhythmias –global longitudinal strain, radial strain and mechanical dispersion as potential markers for ventricular arrhythmias, and most recently, myocardial work as a potential marker for arrhythmia risk.[21]

On the one hand, ventricular arrhythmias might develop as a result of channelopathies, toxicity, or other unknown reasons. On the other hand, in practice, structural heart disease is a quite common cause of ventricular arrhythmias. Echocardiography is applied in the evaluation of structural and functional cardiac problems and, in recent years, to detect patients at high risk of ventricular arrhythmia. Mechanical contraction of the heart is an end product of electrical conduction in the heart and cardiomyocyte depolarization (excitation-contraction coupling).[8] Pathological changes in myocardial activity magnitude and timing lead to changes in electrocardiogram recordings. These slight electrical variations must logically be transferred into mechanical variations. Intraventricular conduction delay and dispersion (QRS) are associated with an increased arrhythmia risk in a number of heart diseases and are commonly assessed by QRS and QT duration.[9] Physiological variability in QRS and QTc intervals is associated with slight but significant changes in myocardial deformation patterns. Better longitudinal stress parameters and increased mechanical dyssynchrony have been associated with longer QTc and increased QRS delay. Integrating the effects of ventricular conduction delay on repolarization, myocardial mechanics, and synchrony is probably going to improve our understanding of electromechanical coupling.[9] The idea of using additional echocardiographic markers for arrhythmogenic burden with electrocardiographic ones has a beneficial practical application. The need of new echocardiographic markers to assess myocardial function and arrhythmogenic burden, and their prognostic value, is the subject of our current review.

Left ventricular echocardiographic parameters for evaluating myocardial function and arrhythmogenic burden and their prognostic value

Strain imaging

Initially, strain imaging was used as a function of tissue Doppler imaging.[22] From three projections: three-cavity, four-cavity and two-cavity, longitudinal, circumferential, and radial deformations, as well as torsion, are quantified.[23] Of the three, longitudinal deformation is the most applicable due to high reproducibility[1,5], but also the subendocardial layers where the longitudinal fibers are located are most vulnerable to ischemia[23]. Longitudinal strain provides the best information on cardiac mechanics, by quantifying GLS, regional strain, mechanical dispersion (MD) and, lately, myocardial work as cardiovascular outcome potential markers, including arrhythmia risk.

Global longitudinal strain of the left ventricle

Data in the literature in recent years demonstrate that GLS can detect left ventricular systolic dysfunction at an earlier stage than the ejection fraction.[24] This fact has its own logical explanation. Ejection fraction is based on volume measurements, while GLS assesses myocardial contractile function. Therefore, the first is an indirect surrogate, and the second a direct surrogate of systolic function. Secondly, GLS reflects the function of the longitudinal fibers that are in the subendocardium, and they are the ones that are most vulnerable to ischemia. It is a fact that coronary perfusion spreads from the subendocardium to the subepicardium. And, last but not least, there is much data that GLS correlates with the size of the infarct area, with myocardial fibrosis and the neurohumoral response.[25,26]

Fig. 1 shows GLS (18% –slightly decreased) in a patient with normal LVEF (62%) and normal MD (44.8 msec). These results suggest more frequent monitoring of these parameters. A decrease in GLS and a prolongation of MD will lead to a change in therapeutic behavior.

At this stage of knowledge, GLS is particularly suitable for patients with normal echocardiographic parameters such as left ventricular ejection fraction, left atrial diameter, left ventricular wall thickness, and aortic root.

Informative value of GLS for ventricular arrhythmias

Data from the literature demonstrate that GLS is prognostic in relation to cardiovascular events, in contrast to ejection fraction and Killip class.[27-29] Despite heterogeneity in endpoints, monitoring and duration of follow-up, GLS predicts ventricular arrhythmias. Moreover, GLS can be used to select candidates suitable for an implantable cardioverter defibrillator (ICD).

At this stage, ventricular arrhythmia and sudden cardiac death risk stratification at an ejection fraction below 35% is considered suboptimal. But at present, it is relied mainly on this fact.[30-32] In a study by Ersbøll et al., the authors es-

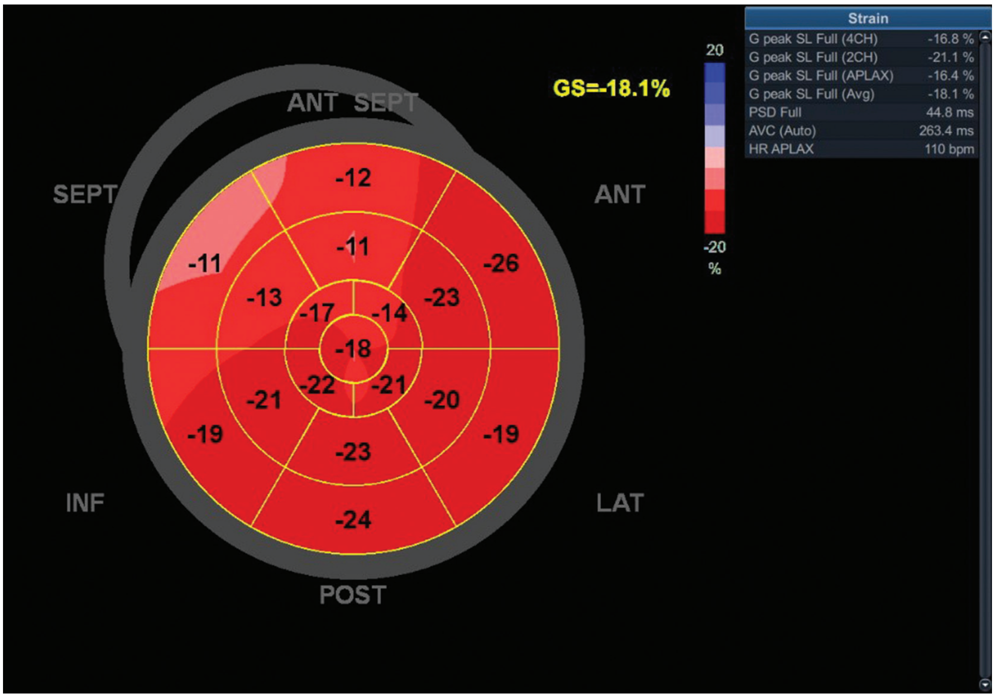

Figure 1. Examples of the longitudinal strain bull's eye plot in a patient with arterial hypertension, a 55-year-old male; metabolic syndrome; basal septal wall thickness is 14 mm, left ventricular posterior wall end diastole is 9 mm, and LVEF is 62%.

tablished the significance of GLS provides important prognostic information in patients with LVEFs >40% above and beyond traditional indexes of high-risk MI.[11]

In a prospective study, Ersbøll et al. state that GLS leads to the determination of the risk of ventricular arrhythmias in myocardial infarction acute phase of up to 48 hours. Both GLS and MD were significantly and independently related to sudden cardiac death/ventricular arrhythmias in these patients with acute myocardial infarction. In this study GLS improved risk stratification above and beyond existing risk factors. GLS was significantly reduced in patients who presented with ventricular arrhythmias compared to patients who did not.[13]

According to data from Haugaa et al., in patients with a prolonged QT interval it was observed that left ventricular mechanical dispersion measured by tissue Doppler imaging was superior to the corrected QT interval.[33] This could better the clinical approach in patients with a prolonged QT interval.

A study by the same team Haugaa et al. in patients after myocardial infarction and ICD, only the combination of MD and GLS can select with greater precision suitable patients for ICD therapy, especially those with an ejection fraction of >35%. The study has multiple limitations of a technical nature.[34]

In patients with a corrected tetralogy of Fallot, Van Grootel et al. discovered that left ventricular as well as right ventricular longitudinal strain add additional information to the routine prognostic risk evaluation of sudden cardiac death and the development of heart failure.[35]

In ischemic heart disease, the role of GLS as a ventricular arrhythmia prognostic marker is controversial. A meta-analysis found that GLS was not associated with ventricular arrhythmias.[16]

Data from a large study such as MADIT-CRT provides strong evidence unlike meta-analyses. In myocardial infarction, there is other data that GLS predicts ventricular arrhythmias, but in a retrospective analysis or in a composite outcome with heart failure or mortality. In a substudy of MADIT-CRT with continuous rhythm monitoring and assessment of ventricular arrhythmias[36], GLS was found to correlate with ventricular arrhythmias in nonischemic cardiomyopathies. Regardless of the small number of patients with nonischemic cardiomyopathy, greater MD was associated with a higher incidence of ventricular tachycardia (VT), ventricular fibrillation (VF).[37]

Data from the beginning of 2024 shows that GLS is a useful indicator in finding suitable patients with hypertrophic cardiomyopathy who are implantable cardioverter defibrillator candidates, confirming the strong link between GLS and fatal arrhythmias and sudden cardiac death.[38]

Mechanical dyssynchrony, mechanical dispersion and ventricular arrhythmia

Nature of mechanical dyssynchrony

Mechanical dyssynchrony represents the inhomogeneity of myocardial contraction within the boundaries of the left ventricle. For several decades, mechanical dyssynchrony has been applied in ventricular arrhythmia prediction in specific patient groups and to identify those suitable for resynchronization therapy. In medical practice, mechanical dispersion is the most commonly used measure of dyssynchrony.[39]

Nature of mechanical dispersion

Generally, in a 16-segment model, MD is defined as the standard deviation of peak longitudinal strain time. MD has excellent reproducibility (intra–and interobserver –0.95 and 0.94).[40] Fibrosis results in a heterogeneous contraction pattern as it leads to electrical dispersion. The latter affects the activation time as well as the refractoriness. MD correlates with fibrosis, therefore correlates with the risk of ventricular arrhythmias. Data on MD in healthy subjects are scarce, and it has mainly been studied to predict ventricular arrhythmias.[41]

Rodríguez-Zenella et al. are the only ones that set themselves the task of determining reference values in healthy people. They studied 334 healthy volunteers (Caucasian race, mean age 54 years, 54% female). They found a total normal value of 34±10 ms with an upper limit of 56 ms. No difference in MD was found between men and women, but it increased with age. The authors found that age, GLS, and E/e' were independently correlated to MD.[40]

Aagaard et al. studied not only healthy individuals but included 2529 participants. They found that ischemic heart disease, hypertension, GLS, e' and left ventricular ejection fraction correlated with higher MD.[28]

Mechanical dispersion and ventricular arrhythmias

The association between MD and ventricular arrhythmias has been widely studied, similarly to GLS. Some patient populations have been studied more thoroughly – those with heart failure[29,42] and ischemic heart disease[14,32], prolonged QT interval syndrome[33] and arrhythmogenic cardiomyopathy[34], also in congenital heart disease[35]. There are two systematic reviews evaluating the potential of MD to predict ventricular arrhythmias.

Kawakami et al. made an ischemic cardiomyopathy meta-analysis, including 12 studies with a total of 3198 patients during 17 to 70 months of follow-up. The analysis showed higher MD in patients with ventricular arrhythmias and independently associated with them, and that MD was better than left ventricular ejection fraction and GLS for prediction of ventricular arrhythmias. A limiting factor is the heterogeneity of the included studies. However, Kawakami et al. discovered that a 1-SD change in LVMD was a stronger predictor of VA events compared with the same changes in LVEF and GLS. The patients with VA events had significantly greater mechanical dispersion compared with those without VA events, with 60 ms being an acceptable cutoff LVMD value for predicting VA events. An interesting claim made by Kawakami et al. is that MD in CRT patients cannot predict ventricular arrhythmias because CRT influences arrhythmia risk and regional timing.[16]

Unlike previous data, Harapoz et al. presented the possibilities of MD in 346 patients with non-ischemic cardiomyopathy in a meta-analysis, finding no significant association between MD and ventricular arrhythmias. A limiting factor is the small number of subjects included. What remains an open question is whether MD is an important ventricular arrhythmia predictor in non-ischemic cardiomyopathy as opposed to ischemic cardiomyopathy, in which pathophysiological mechanisms lead to the spread of fibrosis in the myocardium.[42]

In the MADIT-CRT study of 1064 patients with continuous rhythm monitoring, Biering-Sørensen et al. found that for a mean of 2.9 years, those who had ventricular arrhythmias had similar MD to those without ventricular arrhythmias.[36]

Kutyifa et al. claim in their analysis that baseline MD does not predict ventricular arrhythmias. However, patients with left-bundle branch block CRT who improved their MD after 12 months had a lower risk of ventricular arrhythmias.[39] Van der Bijl et al. drew similar conclusions in a retrospective study of 1185 patients, namely that MD at 6 months predicted ventricular arrhythmias.[32] Almost all relevant studies highlight the important issue that the timing of MD examination is very important when evaluating its prognostic value.

In the MeDiPace transcatheter aortic valve implantation study, MD was considerably higher in patients who required a pacemaker following transcatheter aortic valve implantation compared with patients without a pacemaker. Additionally, MD and right-bundle branch block were independent predictors of pacemaker implantation following transcatheter aortic valve implantation. MD improves prognosis when combined with right-bundle branch block. These results suggest that PM implantation in patients with normal MD should be avoided.[43]

In a 2020 study in the middle-aged general population, the predominant coronary heart disease and hypertension were linked to increased mechanical variance, which probably indicates an increased fatal arrhythmia and sudden cardiac death risk. Although less severe, systolic and diastolic dysfunction are also linked to increased mechanical dispersion.[28]

There is scarce data in the literature on MD in patients with metabolic syndrome, which covers a huge cohort of patients in clinical practice, with the scope of a pandemic.[44] The term "metabolic syndrome" encompasses a group of disorders characterized by central obesity, hypertension, impaired glucose tolerance, and atherogenic dyslipidemia. Insulin resistance and type 2 diabetes cause a variety of functional, metabolic and structural changes leading to myocardial damage and heart failure progression. Echocardiography is a first-line research method for studying cardiac structural and functional changes associated with metabolic syndrome and type 2 diabetes. As the review of literature shows, changes in all indicators of left and right ventricular systolic and diastolic function in metabolic syndrome have not been studied. 2D strain echocardiography can detect early left ventricular dysfunction as well as the asymptomatic stage of metabolic syndrome. The latter affects the functional state of the heart by causing a decrease in GLS. In the case of normal GLS in metabolic syndrome, regional strain is reduced. GLS reference limits in metabolic syndrome patients have not been established. Data on the role of MD as a ventricular arrhythmia predictor in metabolic syndrome is scarce.[45]

Regional strain of the left ventricle

GLS demonstrates the global LV deformation, the assessment of the tissue deformation in certain areas of the left ventricle is done by regional strain. Through the gold standard examination of the myocardium, it is established that longitudinal and radial deformation are progressively impaired by non-, peri- and infarct zones.[32]

In the VALIANT study, regional strain was found to be impaired in areas that were assessed as normokinetic. On the other hand, a greater number of segments with impaired regional strain is associated with higher mortality and heart failure.[46]

Biering-Sørensen et al. reveal a very important clinical fact that reduced regional strain outside the area of the culprit artery demonstrates limited reserve after myocardial infarction.[20] There is evidence that regional strain is associated with a greater likelihood of left ventricular thrombus formation. A limiting factor for regional strain compared to GLS is its poorer reproducibility and heterogeneity.[7]

Informative value of regional strain for ventricular arrhythmias

In myocardial infarction patients, the presence of fibrous tissue in the myocardial zone is the substrate with arrhythmogenic potential. At the same time, the peri-infarct zone has intermediate non-transmural fibrosis, which leads to electrical dispersion, delayed conduction, and one-way block. The latter produce substrates for ventricular arrhythmias.[47]

There is scarce data in the literature on the informative value of regional strain in relation to ventricular arrhythmias than GLS and MD. Nguyen et al. studied the potential of regional strain to predict ICD in 424 ischemic cardiomyopathy and a prophylactic implanted cardioverter defibrillator patients. It was found that regional strain in the peri-infarct zone was independently related to the need for an ICD.[17] Hoogslag et al. studied the informativeness of regional strain for predicting proper treatment with ICD or cardiac mortality by echocardiography at baseline and at 3 months in 79 patients with myocardial infarction. No difference was found in regional strain identified in infarct, peri-infarct or remote area at baseline between patients.[48] In a 25-month follow-up of 51 patients with documented ventricular arrhythmias or 24-hour Holter monitoring, recording at hospitalization or electrophysiological examination. Data shows that longitudinal and circumferential strain demonstrate high AUCs for detecting ventricular arrhythmias. Furthermore, only abnormal circumferential peri-infarct strain and MD were independently related to ventricular arrhythmias. More massive myocardial infarctions are not limited to subendocardial ischemia, reflected in GLS, but also reduce circumferential strain.[37]

In heart failure patients, MADIT-CRT is the largest study to investigate the potential of regional longitudinal strain and its role in predicting ventricular arrhythmias.[36]

Biering-Sørensen et al. studied 1064 patients and found that longitudinal strain in the lower and posterior segments of the left ventricular wall correlated with the occurrence of ventricular tachycardia/flutter.[20] A study from Denmark with 401 patients demonstrated the association between inferior wall deformation and ventricular arrhythmias.[24] One of the hangings is differentiation of the parasympathetic and sympathetic innervation of the left ventricle and differential deformation of the wall.[25]

Data from the literature verify the gold standard of cardiac MRI to anatomically detect areas of dysfunctional myocardium and predict ventricular arrhythmias and mortality.[49]

Guidelines for future research on strain measurements

Although GLS, MD, and regional strain are the most widely studied strain indicators in terms of predicting ventricular arrhythmias, theoretically any other indicators of myocardial mechanics may have prognostic value. The latter include indicators of paradoxical movement and myocardial work. However, currently, their associations with ventricular arrhythmias have been poorly studied.

Paradoxical motion is a change in myocardial tissue motion, with left ventricular segments lengthening when they should be shortening. In clinical practice, it most often appears as early systolic lengthening or as postsystolic shortening. Paradoxical motion is most commonly observed in ischemic heart disease and shows ischemic segments with potential tissue viability. Nevertheless, they can also occur in other conditions, including conduction abnormalities and mitral annular disjunction. It should also be mentioned that to some extent, they may occur under regular circumstances as part of normal physiology. These indices of myocardial deformation are highly correlated with ischemia and possibly with the extent of myocardial involvement. It is logical that they are also markers for an increased risk of ventricular arrhythmias. But this claim needs to be proven.[50]

In a study of 569 patients, Haugaa et al. found that patients who developed ventricular arrhythmias had a higher degree of postsystolic shortening, (postsystolic stress index), compared to patients without ventricular arrhythmias. All patients had a left ventricular ejection fraction >35%. Postsystolic strain index was not an independent predictor of ventricular arrhythmias when MD was included.[33] Groeneveld et al. also demonstrated a more frequent and widespread postsystolic shortening in patients with idiopathic ventricular arrhythmias.[51]

The latest trend in strain development is a new method for assessing myocardial performance with pressure-strain loop analysis. This method, introduced by Russell et al. in 2012, enables assessment of the myocardial performance by combining data on strain, arterial blood pressure, and valvular event time. This method has excellent reproducibility. Inclusion of arterial pressure as an afterload assessment assesses systolic function in more detail.[52]

Practical approaches to measure myocardial performance are described in detail in the literature. A significant point in the measurement is the information obtained about the constructive work, wasted work and work efficiency. All three are affected by the paradoxicality of motion. And the latter can point us to the risk of ventricular arrhythmias.[53]

In a meta-analysis, Truong et al. provide echocardiographic reference ranges for noninvasive indices of myocardial work. As reference for clinical and research use these normal values could serve. Echocardiographic reference ranges for indices of myocardial work were established. Gender significantly contributed to variations in normal values of myocardial work. Myocardial work indices had excellent and good intra- and interobserver reproducibility.[54] It is yet to be proven whether or not evaluating myocardial work has a clinical relevance.

At this stage, it is assumed that myocardial performance provides clinical information superior to GLS. This is what the literature shows. A case report by Jaworski et al. discusses the possible application of regional work in a patient with ventricular fibrillation.[55] In a study of hypertrophic cardiomyopathy in 110 patients, Hiemstra et al. found constructive work to be a predictor of a composite clinical endpoint that included sudden cardiac death and ICD therapy, which was found in 11 of 24 cases.[56]

Currently, there is no data on the relationship between myocardial performance and ventricular arrhythmia risk. This question remains to be clarified in additional clinical studies.

CONCLUSION

Data on the relationship of strain assessment (GLS and MD) and ventricular arrhythmias in a large spectrum of cardiovascular diseases demonstrate likely clinical utility. At this stage, there is a lack of studies that evaluate whether or not a patient is suitable for an implantable cardioverter defibrillator by randomizing based on strain measurement. The new methods –paradoxical movement and myocardial work are far from being a prognostic factor. Much more research is necessary in this direction, but the need for the clinician to support these methodologies is likely to give impetus to such research.

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Competing Interests

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REFERENCES

1. Karlsen S, Dahlslett T, Grenne B, et al. Global longitudinal strain is a more reproducible measure of left ventricular function than ejection fraction regardless of echocardiographic training. Cardiovasc Ultrasound 2019; 17:18.
2. Stecker EC, Vickers C, Waltz J, et al. Population-based analysis of sudden cardiac death with and without left ventricular systolic dysfunction: two-year findings from the Oregon Sudden Unexpected Death Study. J Am Coll Cardiol 2006; 47(6):1161–6.
3. Goldberger JJ, Subačius H, Patel T, et al. Sudden cardiac death risk stratification in patients with nonischemic dilated cardiomyopathy. J Am Coll Cardiol 2014; 63(18):1879–89.
4. Vaduganathan M, Patel RB, Michel A, et al. Mode of death in heart failure with preserved ejection fraction. J Am Coll Cardiol 2017; 69(5):556–69.
5. Pfeiffer ER, Tangney JR, Omens JH, et al. Biomechanics of cardiac electromechanical coupling and mechanoelectric feedback. J Biomech Eng 2014; 136(2):021007.
6. Verdugo-Marchese M, Coiro S, Selton-Suty C, et al. Left ventricular myocardial deformation pattern, mechanical dispersion, and their relations with electrocardiogram markers in the large population-based STANISLAS cohort: insights into electromechanical coupling. Eur Heart J Cardiovasc Imaging 2020; 21(11):1237–45.
7. Bjerregaard CL, Skaarup KG, Lassen MCH, et al. Strain imaging and ventricular arrhythmia. Diagnostics (Basel) 2023; 13(10):1778.
8. Negishi K, Negishi T, Kurosawa K, et al. Practical guidance in echocardiographic assessment of global longitudinal strain. JACC Cardiovasc Imaging 2015; 8:489–92.
9. DeVore AD, McNulty S, Alenezi F, et al. Impaired left ventricular global longitudinal strain in patients with heart failure with preserved ejection fraction: Insights from the RELAX trial. Eur J Heart Fail 2017; 19:893–900.
10. Munk K, Andersen NH, Nielsen SS, et al. Global longitudinal strain by speckle tracking for infarct size estimation. Eur J Echocardiogr 2011; 12:156–65.
11. Ersbøll M, Valeur N, Mogensen UM, et al. Prediction of all-cause mortality and heart failure admissions from global left ventricular longitudinal strain in patients with acute myocardial infarction and preserved left ventricular ejection fraction. J Am Coll Cardiol 2013; 61:2365–73.
12. Santangeli P, Dello Russo A, Casella M, et al. Left ventricular ejection fraction for the risk stratification of sudden cardiac death: Friend or foe? Intern Med J 2011; 41:55–60.
13. Ersbøll M, Valeur N, Andersen MJ, et al. Early echocardiographic deformation analysis for the prediction of sudden cardiac death and life-threatening arrhythmias after myocardial infarction. JACC Cardiovasc Imaging 2013; 6:851–60.
14. Haugaa KH, Grenne BL, Eek CH, et al. Strain echocardiography improves risk prediction of ventricular arrhythmias after myocardial infarction. JACC Cardiovasc Imaging 2013; 6:841–50.
15. Haugaa KH, Smedsrud MK, Steen T, et al. Mechanical dispersion assessed by myocardial strain in patients after myocardial infarction for risk prediction of ventricular arrhythmia. JACC Cardiovasc Imaging 2010; 3:247–56.
16. Kawakami H, Nerlekar N, Haugaa KH, et al. Prediction of ventricular arrhythmias with left ventricular mechanical dispersion. JACC Cardiovasc Imaging 2020; 13:562–72.

17. Nguyen BL, Capotosto L, Persi A, et al. Global and regional left ventricular strain indices in post-myocardial infarction patients with ventricular arrhythmias and moderately abnormal ejection fraction. Ultrasound Med Biol 2015; 41:407–17.
18. Yu S, Zhou J, Yang K, et al. Correlation of myocardial strain and late gadolinium enhancement by cardiac magnetic resonance after a first anterior ST-segment elevation myocardial infarction. Front Cardiovasc Med 2021; 8:705487.
19. Wang N, Hung CL, Shin SH, et al. Regional cardiac dysfunction and outcome in patients with left ventricular dysfunction, heart failure, or both after myocardial infarction. Eur Heart J 2016; 37:466–72.
20. Biering-Sørensen T, Jensen JS, Pedersen SH, et al. Regional longitudinal myocardial deformation provides incremental prognostic information in patients with ST-segment elevation myocardial infarction. PLoS One 2016; 11:e0158280.
21. Amzulescu MS, Langet H, Saloux E, et al. Head-to-head comparison of global and regional two-dimensional speckle tracking strain versus cardiac magnetic resonance tagging in a multicenter validation study. Circ Cardiovasc Imaging 2017; 10:e006530.
22. Verma A, Marrouche NF, Schweikert RA, et al. Relationship between successful ablation sites and the scar border zone defined by substrate mapping for ventricular tachycardia post-myocardial infarction. J Cardiovasc Electrophysiol 2005; 16:465–71.
23. Hoogslag GE, Thijssen J, Höke U, et al. Prognostic implications of left ventricular regional function heterogeneity assessed with two-dimensional speckle tracking in patients with ST-segment elevation myocardial infarction and depressed left ventricular ejection fraction. Heart Vessel 2014; 29:619–28.
24. Winsløw U, Elming M, Thune JJ, et al. Reduced inferior wall longitudinal strain is associated with malignant arrhythmias in non-ischemic heart failure. Pacing Clin Electrophysiol 2023; 46(7):721–8.
25. Sasano T, Abraham MR, Chang KC, et al. Abnormal sympathetic innervation of viable myocardium and the substrate of ventricular tachycardia after myocardial infarction. J Am Coll Cardiol 2008; 51:2266–75.
26. Tayal B, Gorcsan J, Delgado-Montero A, et al. Mechanical dyssynchrony by tissue Doppler cross-correlation is associated with risk for complex ventricular arrhythmias after cardiac resynchronization therapy. J Am Soc Echocardiogr 2015; 28:1474–81.
27. Rodríguez-Zanella H, Haugaa K, Boccalini F, et al. Physiological determinants of left ventricular mechanical dispersion: a 2-dimensional speckle tracking echocardiographic study in healthy volunteers. JACC Cardiovasc Imaging 2018; 11:650–1.
28. Aagaard EN, Kvisvik B, Pervez MO, et al. Left ventricular mechanical dispersion in a general population: Data from the Akershus cardiac examination 1950 study. Eur Heart J Cardiovasc Imaging 2020; 21:183–90.
29. Mornos C, Muntean D, Mornos A, et al. Risk stratification in patients with heart failure: The value of considering both global longitudinal left ventricular strain and mechanical dispersion. Can J Physiol Pharmacol 2017; 95:1360–8.
30. Matsuzoe H, Tanaka H, Matsumoto K, et al. Left ventricular dyssynergy and dispersion as determinant factors of fatal ventricular arrhythmias in patients with mildly reduced ejection fraction. Eur Heart J Cardiovasc Imaging 2016; 17:334–42.
31. Banasik G, Segiet O, Elwart M, et al. LV mechanical dispersion as a predictor of ventricular arrhythmia in patients with advanced systolic heart failure: A pilot study. Herz 2016; 41:599–604.
32. Van der Bijl P, Khidir MJH, Leung M, et al. Reduced left ventricular mechanical dispersion at 6 months follow-up after cardiac resynchronization therapy is associated with superior long-term outcome. Heart Rhythm 2018; 15:1683–9.
33. Haugaa KH, Edvardsen T, Leren TP, et al. Left ventricular mechanical dispersion by tissue Doppler imaging: A novel approach for identifying high-risk individuals with long QT syndrome. Eur Heart J 2009; 30:330–7.
34. Lie ØH, Rootwelt-Norberg C, Dejgaard LA, et al. Prediction of life-threatening ventricular arrhythmia in patients with arrhythmogenic cardiomyopathy: a primary prevention cohort study. JACC Cardiovasc Imaging 2018; 11:1377–86.
35. Van Grootel RWJ, Van den Bosch AE, Baggen VJM, et al. The prognostic value of myocardial deformation in adult patients with corrected tetralogy of Fallot. J Am Soc Echocardiogr 2019; 32:866–75.e2.
36. Biering-Sørensen T, Knappe D, Pouleur AC, et al. Regional longitudinal deformation improves prediction of ventricular tachyarrhythmias in patients with heart failure with reduced ejection fraction: a MADIT-CRT substudy (Multicenter Automatic Defibrillator Implantation Trial-Cardiac Resynchronization Therapy). Circ Cardiovasc Imaging 2017; 10(1):e005096.
37. Kosiuk J, Dinov B, Bollmann A, et al. Association between ventricular arrhythmias and myocardial mechanical dispersion assessed by strain analysis in patients with nonischemic cardiomyopathy. Clin Res Cardiol 2015; 104:1072–7.
38. Nikoo MH, Zarrabi M, Moaref A, et al. Global longitudinal strain may be the one that appropriately identifies candidates of ICD implantation. Cardiol Res Pract 2024; 16:2214072.
39. Kutyifa V, Pouleur AC, Knappe D, et al. Dyssynchrony and the risk of ventricular arrhythmias. JACC Cardiovasc Imaging 2013; 6:432–44.
40. Rodríguez-Zanella H, Haugaa K, Boccalini F, et al. Physiological determinants of left ventricular mechanical dispersion: a 2-dimensional speckle tracking echocardiographic study in healthy volunteers. JACC Cardiovasc Imaging 2018; 11:650–1.
41. Vassallo JA, Cassidy DM, Kindwall KE, et al. Nonuniform recovery of excitability in the left ventricle. Circulation 1988; 78:1365–72.
42. Harapoz M, Zada M, Matthews J, et al. Echocardiographic predictors of ventricular arrhythmias in patients with non-ischemic cardiomyopathy. Int J Cardiol Heart Vasc 2022; 39:100962.
43. Kaya E, Andresen K, Lie ØH, et al. Left ventricular mechanical dispersion as a predictor of the need for pacemaker implantation after transcatheter aortic valve implantation: MeDiPace TAVI study. Eur Heart J Cardiovasc Imaging 2024; 25(4):539–47.
44. Aryee EK, Ozkan B, Ndumele CE. Heart failure and obesity: the latest pandemic. Prog Cardiovasc Dis 2023; 78:43–8.
45. Maisuradze D. Global longitudinal strain in patients with metabolic syndrome and normal ejection fraction. J Cardiol Res Reviews & Reports 2022; SRC/JCRRR-166.
46. Torres WM, Jacobs J, Doviak H, et al. Regional and temporal changes in left ventricular strain and stiffness in a porcine model of myocardial infarction. Am J Physiol Heart Circ Physiol 2018; 315(4):H958–H967.
47. Luisi GA, Pestelli G, Lorenzoni G, et al. Severe impairment of left ventricular regional strain in STEMI patients is associated with post-infarct remodeling. J Clin Med 2022; 11(18):5348.
48. Hoogslag GE, Thijssen J, Höke U, et al. Prognostic implications of left ventricular regional function heterogeneity assessed with two-dimensional speckle tracking in patients with ST-segment elevation

myocardial infarction and depressed left ventricular ejection fraction. Heart Vessel 2014; 29:619–28.

49. Yan AT, Shayne AJ, Brown KA, et al. Characterization of the peri-infarct zone by contrast-enhanced cardiac magnetic resonance imaging is a powerful predictor of post-myocardial infarction mortality. Circulation 2006; 114:32–9.
50. Brainin P. Myocardial postsystolic shortening and early systolic lengthening: current status and future directions. Diagnostics 2021; 11:1428.
51. Smiseth OA, Donal E, Penicka M, et al. How to measure left ventricular myocardial work by pressure-strain loops. Eur Heart J Cardiovasc Imaging 2021; 22:259–61.
52. Russell K, Eriksen M, Aaberge L, et al. A novel clinical method for quantification of regional left ventricular pressure-strain loop area: A non-invasive index of myocardial work. Eur Heart J 2012; 33:724–33.
53. Olsen FJ, Skaarup KG, Lassen MCH, et al. Normal values for myocardial work indices derived from pressure-strain loop analyses: from the CCHS. Circ Cardiovasc Imaging 2022; 15:e013712.
54. Truong VT, Vo HQ, Ngo TNM, et al. Normal ranges of global left ventricular myocardial work indices in adults: a meta-Analysis. J Am Soc Echocardiogr 2022; 35:369–77.
55. Jaworski K, Firek B, Syska P, et al. Malignant arrhythmia associated with mitral annular disjunction: Myocardial work as a potential tool in the search for a substrate. Kardiol Pol 2022; 80:93–4.
56. Hiemstra YL, Van der Bijl P, El Mahdiui M, et al. Myocardial work in nonobstructive hypertrophic cardiomyopathy: implications for outcome. J Am Soc Echocardiogr 2020; 33:1201–8.

Обзор альтернативных измерений в визуализации продольной деформации для прогнозирования желудочковой аритмии

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Резюме

Глобальная продольная деформация была установлена как надёжный инструмент для оценки глобальной функции левого желудочка и маркер субклинической дисфункции левого желудочка, нераспознаваемой фракцией выброса. С другой стороны, желудочковые аритмии являются наиболее распространённой причиной внезапной сердечной смерти. Их раннее выявление является сложной задачей. Возможные прогностические маркеры риска желудочковых аритмий обсуждаются в литературе – электрокардиографические, магнитно-резонансные, компьютерные томографии, радионуклидные визуализации и маркеры новых эхокардиографических методов. Из последних на данном этапе знаний несколько маркеров обсуждались как информативные для прогнозирования желудочковых аритмий – глобальная продольная деформация, радиальная деформация и механическая дисперсия, а совсем недавно – работа миокарда. Насколько нам известно, глобальная продольная деформация особенно полезна у пациентов с нормальными эхокардиографическими параметрами, такими как фракция выброса левого желудочка, диаметр левого предсердия, толщина стенки левого желудочка и корень аорты. Связь между механической дисперсией и желудочковыми аритмиями широко изучалась. Связь изучалась больше в некоторых группах пациентов – сердечная недостаточность, ишемическая болезнь сердца, синдром удлинённого интервала QT и аритмогенная кардиомиопатия, врождённый порок сердца. Роль механической дисперсии как предиктора желудочковых аритмий при метаболическом синдроме незначительна.

Ключевые слова

фракция выброса, глобальная продольная деформация, механическая дисперсия, работа миокарда, радиальная деформация