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Possibilities and prospects of echocardiographic diagnostics of regional contractility disorders of the left ventricular myocardium in patients with chronic ischemic heart disease

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Abstract

Currently, the primary method for identifying transient disorders of local contractility of the left ventricular myocardium in patients with coronary atherosclerosis remains visual assessment of myocardial contractility under physical or pharmacological stress testing. Visual assessment of myocardial contractility, especially in stress tests, requires extensive experience in conducting such studies. However, visual assessment by even the most experienced operator remains subjective. Consequently, a principal focus in diagnosing left ventricular regional wall motion abnormalities has been, and remains, the development of methods for objective quantitative assessment of functional status across different left ventricular myocardial segments. A significant success in this area was the development of speckle-tracking echocardiography technique, which allows for a quantitative assessment of myocardial deformation during its contraction and relaxation.

The review presents the results of studies indicating that the determination of left ventricular myocardial deformation indices may become an alternative to the traditional method, devoid of such disadvantages as the subjectivity of visual information perception and very high requirements for the operator's qualification level. Deepening knowledge about the mechanisms, clinical significance of various myocardial deformation indices, the improvement of both the speckle-tracking echocardiography technique itself and the algorithms of automated processing of data creates a real prospect for its introduction into clinical practice as the main method for identifying transient disorders of local left ventricular contractility in patients with hemodynamically significant coronary atherosclerosis.

Keywords: chronic ischemic heart disease, stress-echocardiography, speckle tracking echocardiography technology, regional myocardial contractility disorders, longitudinal systolic deformation of the left ventricle, myocardial postsystolic shortening.

Conflict of interest: nothing to disclose.

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Возможности и перспективы эхокардиографической диагностики нарушений локальной сократимости миокарда левого желудочка при хронической ишемической болезни сердца

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Аннотация

В настоящее время основным методом выявления транзиторных нарушений локальной сократимости миокарда левого желудочка у больных с коронарным атеросклерозом служит визуальная оценка сократимости миокарда при физической или фармакологической нагрузке. Визуальная оценка сократительной способности миокарда, особенно в нагрузочных пробах, требует большого опыта в проведении такого рода исследований. Однако зрительная оценка даже самого опытного оператора по-прежнему не лишена субъективизма. В связи с этим одним из магистральных направлений диагностики нарушений локальной сократимости левого желудочка была и остается разработка методов объективной количественной оценки функционального состояния различных участков миокарда левого желудочка. Существенным успехом на этом пути стало создание методики отслеживания серого пятна (speckle-tracking эхокардиография), позволяющей получить количественную оценку деформации миокарда при его сокращении и расслаблении.

В обзоре представлены результаты исследований, свидетельствующие, что определение показателей деформации миокарда левого желудочка может стать альтернативой традиционному методу, лишенной таких его недостатков, как субъективизм восприятия визуальной информации и очень высокие требования к уровню квалификации оператора. Углубление знаний о механизмах и клиническом значении различных показателей деформации миокарда, наряду с совершенствованием как самой методики speckle-tracking эхокардиографии, так и алгоритмов автоматизированной обработки получаемых с ее помощью данных, создает реальную перспективу ее внедрения в клиническую практику в качестве основного метода выявления транзиторных нарушений локальной сократимости миокарда левого желудочка у больных с гемодинамически значимым коронарным атеросклерозом.

Ключевые слова: хроническая ишемическая болезнь сердца, стресс-эхокардиография, нарушения локальной сократимости миокарда, технология speckle tracking echocardiography, продольная систолическая деформация левого желудочка, постсистолическое укорочение миокарда.

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Список сокращений
ГПС – глобальный продольный стрейн; ИБС – ишемическая болезнь сердца; КАГ – коронарная ангиография; НЛС – нарушение локальной сократимости; ОВ – огибающая ветвь; ПКА – правая коронарная артерия; ПМЖВ – передняя межжелудочковая ветвь; ПСИ – постсистолический индекс; ПСУ – постсистолическое укорочение; ТИМ – транзиторная ишемия миокарда; ЭхоКГ – эхокардиография; AUC – площадь под кривой; РПС – региональный продольный стрейн.

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INTRODUCTION

One of the principal tasks of echocardiographic examination (EchoCG) is to identify disorders of regional contractility (RCD) of the left ventricle myocardium that may be caused by a number of cardiovascular diseases [1]. The most common cause of RCD is the coronary heart disease (CHD), specifically, the ischemic necrosis (infarction) of the myocardium followed by replacement of the necrotized section of the myocardium with connective tissue. The reason for deterioration of the myocardial contractility may be not only necrosis but also the persistent reduction of blood supply to the section of the myocardium to the level sufficient to maintain the viability of myocardiocytes, but insufficient for them to perform their contractile function (hibernating myocardium) [2]. Considerably less frequently, ILC stems from other causes, e.g. amyloid deposition [3].

In the above mentioned cases, RCDs are persistent, i.e. present over a long period regardless of the work performed by the heart. The reason for transient RCDs is the transitory myocardial ischemia (TMI), most frequently arising out of impossibility of adequate blood supply of the myocardium under the increasing cardiac load. The morphological substrate of TMI is typically hemodynamically significant atherosclerosis of the coronary arteries. At the same time, restricted myocardial blood supply under stress may also result from compression of the coronary arteries (so-called “myocardial bridging”) or from the loss of the ability of coronary arteries to dilate in response to increased blood flow velocity (Syndrome X). The onset of TMI at rest might stem from a spasm of the coronary artery (vasospastic angina) [4]. Thus, identification of RCDs is only the first step of the diagnostic process whose task is the identification of causes of their onset.

This review article provides an analysis of the modern possibilities offered by EchoCG in the diagnostics and differential diagnostics of persistent and transient RCDs of the left ventricle myocardium.

Methodology of search for sources. The search for literature was performed in the Russian Science Citation Index (RSCI) and PubMed databases using the following keywords: speckle tracking echocardiography, stress-echocardiography and regional myocardial contractility disorders, or global longitudinal strain of the left ventricle, or myocardial postsystolic shortening. The total number of analyzed papers was 3215. The conditions for the selection of journal articles and other materials were as follows: year of publication: not earlier than 2010; focus on the use of speckle tracking echocardiography during stress tests (stress-echocardiography). The final analysis included 36 selected articles.

VISUAL DIAGNOSTICS OF RCD

From the advent of EchoCG into clinical practice to the present, the major method of identifying RCDs is the visual comparison of contractile activity of various regions of the left ventricular myocardium [1]. Myocardial contractions come with an obvious thickening of the walls of the left ventricle and their shift to the ‘axis line’ of the left ventricle.

In the systolic period, the thickness of the interventricular septum and of the posterior wall of the left ventricle increases by more than 50%, and its transverse size decreases by at least 10% [5]. Visually, it is possible to ascertain a decrease of contractile activity of a certain myocardial region (hypokinesia), complete absence of contractility (akinesia), as well as systolic or systolic-diastolic protrusion of a

region of the myocardium (dyskinesia or aneurysm). While RCD may be present in some regions of the myocardium, increased contractile activity (hyperkinesia) may be observed in other regions. Contractile activity of various regions of the myocardium is often expressed in points: hyperkinesia – 0, normokinesis – 1, hypokinesia – 2, akinesia – 3, dyskinesia – 4, aneurysm – 5 [1].

In order to localize the identified changes, three diagrams of segmental division of the left ventricle are proposed: 16-segment, 17-segment and 18-segment. The 17-segment diagram is now the most widely used, with 6 segments on the basal level, 6 on the medial, and 5 on the apical (**Fig. 1**).

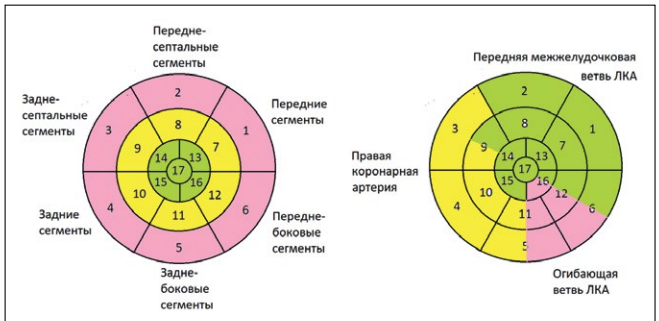
On the same figure, the scheme of blood supply to the left ventricle is shown, proposed by M.D Cerqueira *et al*. (2002) [6]. Depending on the specific features of coronary anatomy, the 9th segment may receive blood from the anterior interventricular branch (*Ramus interventricularis anterior,* RIVA) or the right coronary artery (RCA), 6th, 12th and 16th from the RIVA or from the circumflex branch (CB), and the 5th and 11th from the CB or RCA. Mismatch between RCD and the perfusion territory of a specific coronary artery suggests a non-ischemic etiology, such as deposition of foreign material in the affected myocardial segment.

In order to identify transient RCDs, tests under physical or pharmacological stress are performed under EchoCG control (stress-EchoCG). Exercise stress testing utilizes a cycle ergometer or treadmill, while pharmacological stress testing employs dobutamine infusion. In either case, heart rate increases as does the myocardium demand of oxygen, which, in the event of impossibility of adequate increase of coronary perfusion, results in the onset of TMI and transient RCDs. Modern ultrasonic devices are equipped with a so-called stress system that simultaneously displays the recording of the systolic part of the cardiac cycle at rest and under stress, which significantly facilitates identification of transient RCDs.

Exercise stress testing on an upright (seated) cycle ergometer and especially on a treadmill provides good modeling of actual conditions leading to the onset of TMI but does not allow visual tracking of the contractile activity of the myocardium under stress. Such a possibility arises when the supine (recumbent) cycle ergometer is used; however, due to the non-physiologic nature of the load, it had not become widely employed.

The dobutamine infusion test is less physiologic but provides ideal conditions for a continuous visual assessment of the contractile capacity of the myocardium. At the same time, low-dosage dobutamine infusion test allows identification of the hibernating (viable but not contracting) myocardium. Dobutamine stimulation causes such myocardium to contract, which confirms its viability. Identification of the hibernating myocardium is highly important for the decision-making as to feasibility of revascularization after an infarction [2, 5].

It is to be emphasized that a visual assessment of the contractile capacity of the myocardium, especially in stress tests, requires vast experience in making such examinations. It is officially considered that the results of 50 first examinations performed by an aspiring operator have no diagnostic value [5]. At the same time, an assessment performed even by a highly experienced operator remains subjective, which is



Anterior septal segments Posterior septal segments Anterior segments Posterior segments Anterior lateral segments Posterior lateral segments	Anterior interventricular branch of the LCA Right coronary artery Circumflex branch of the LCA

Figure 1. *Diagram of segmental division and blood supply of the segments of the left ventricle. On the left is a diagram of the segmental division of the left ventricle (pink: basal level, yellow: medial level, green: apical level). On the right: diagram of blood supply to the left ventricle (LCA: left coronary artery).*

Рисунок 1. *Схема сегментарного деления и кровоснабжения сегментов левого желудочка. Слева – схема сегментарного деления левого желудочка (розовый цвет – базальный уровень, желтый – медиальный, зеленый – апикальный). Справа – схема кровоснабжения левого желудочка (ЛКА – левая коронарная артерия).*

confirmed by characteristics of the stress-EchoCG as a predictor of identification of hemodynamically significant coronary atherosclerosis. According to the data from the leading medical centers, the sensitivity and specificity of the stress test is approx. 85–90%, i.e. one in every ten cases of identification of RCD, and one in every ten cases when RCD is deemed absent under stress, are erroneous [5]. Therefore, one of the major trends in the development of EchoCG has been, and remains, the work on methods of objective assessment of contractile capacity of different regions of the left ventricular myocardium. A significant achievement in this area was the development of the speckle-tracking echocardiography that provides quantitative assessment of the myocardium deformation in its contraction and relaxation [7–10].

LONGITUDINAL SYSTOLIC MYOCARDIAL DEFORMATION

During systole, the left ventricle shortens, contracts, and twists along its longitudinal axis, resulting in longitudinal, radial, and circumferential deformation of each myocardial segment. The myocardial deformation comes with a change in the distance between its neighboring points, which enables quantitative assessment of the deformation by comparing the distance between the points in the beginning and the end of systole. These measurements can be obtained using various methods, such as tissue Doppler imaging. However, speckle-tracking echocardiography, a gray-scale pattern tracking technology, is currently considered the gold standard for assessing myocardial deformation. This technology allows for an assessment of all three components of systolic deformation whose diagnostic value may vary in different clinical situations [11]. However, only the longitudinal systolic deformation is recommended for use in present-day clinical practice [12]. This review focuses only on this type of myocardial deformation,

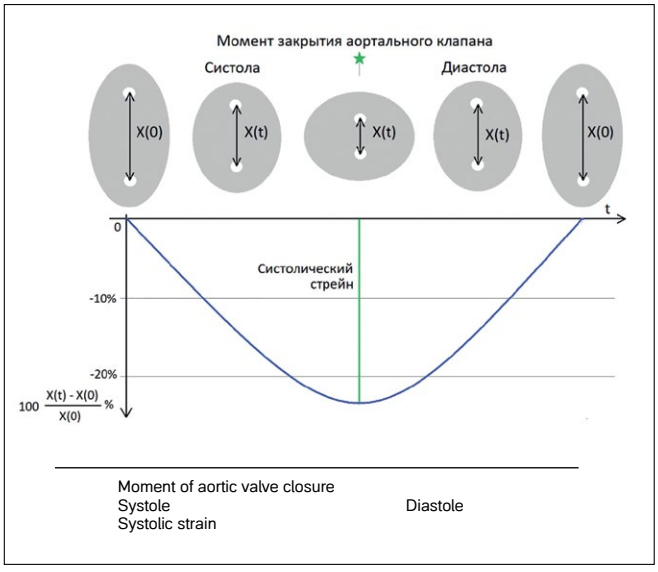


Figure 2. *Changes in the distance between adjacent points of the myocardium and the longitudinal systolic strain index (longitudinal systolic strain) throughout the cardiac cycle (diagram).*

Рисунок 2. *Изменение расстояния между близлежащими точками миокарда и показателя продольной систолической деформации (продольного систолического стрейна) на протяжении кардиоцикла (схема).*

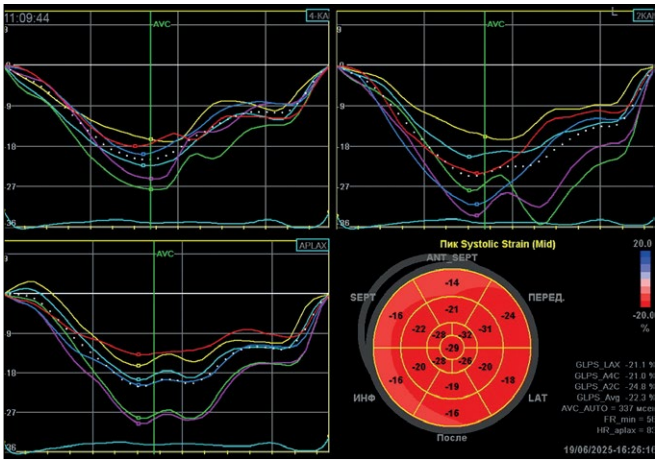


Figure 3. *Results of measuring the longitudinal systolic strain of the left ventricle using the speckle-tracking echocardiography method. Graphs of strain changes throughout the cardiac cycle in all 17 segments of the left ventricle are presented. Strain values at the moment of aortic valve closure are shown on the color map.*

Рисунок 3. *Результаты измерения продольного систолического стрейна левого желудочка методом speckle-tracking эхокардиографии. Представлены графики изменения стрейна на протяжении кардиоцикла во всех 17 сегментах левого желудочка. Значения стрейна в момент закрытия аортального клапана отражены на цветовой карте.*

and the term systolic deformation (systolic strain) shall mean the longitudinal systolic deformation (longitudinal systolic strain).

Speckle-tracking technology allows for making a graph of changes in the distance between the two neighboring points of the myocardium during the cardiac cycle (**Fig. 2**). This distance reaches its maximum in the end of the diastole, when the myocardium is fully relaxed. During the systole, the distance between the tracked points decreases, and upon the end of contraction, it starts increasing. At the same time, the difference between the current and the initial distance during the systole increases, and decreases during the diastole. The percent ratio of this difference at the moment of closure of the aortic valve to the initial distance is used for the qualitative assessment of the systolic myocardial deformation (strain).

During myocardial contraction, the distance between the tracked points decreases, and so the systolic strain is a negative value. However, in the description and statistical processing of the results, the absolute value of the strain is used: the greater the contraction of the myocardium, the greater the value.

The graph of myocardial deformation during the cardiac cycle is created for each of the 17 segments of the left ventricle, and the values of segmental strain at the moment of closure of aortic valve are shown on a color map commonly referred to as the 'bull's eye' (**Fig. 3**). Transition from intense red to intense blue marks the decrease of contractive capacity of the myocardium, from hyperkinesia to dyskinesia.

The color map gives a visual representation of the contractive capacity of various regions of the left ventricular myocardium, for which reason it is often used in educational materials and descriptions of clinical cases. For a generalized assessment of the map data, some indicators are used that represent the contractive capacity of the myocardial segments belonging to a specific region (regional longitudinal strain, RLS). In most cases, regional indicators are calculated as the average value of strain in segments of that region. Specifically, it is shown that in healthy individuals at rest the average strain value in the basal segments is lower than in the apical, while at stress these differences become more pronounced [13].

Studying of the relation of RCD with myocardial ischemia usually involves calculation of average values of longitudinal strain in the regions belonging to perfusion areas of some or other coronary artery (Fig. 1, right). This approach was used in the study of M.K. Smedsrud *et al.* (2012) [14] that included 86 patients with recurrent chest pains. The study did not include patients with prior verified CHD. Speckle-tracking echocardiography at rest was performed before the coronary angiography (CAG), the results of which allowed identification of patients with and without hemodynamically significant coronary stenosis (>50%). It was found that the average values of RLS in patients with hemodynamically significant stenosis were in average 2.2% lower than in the alternative group (17.9±3.5% vs. 20.1±2.9%, p = 0.015). At the same time, the predictive capacity of decreased RLS with respect to identification of hemodynamically significant stenosis by CAG was rather low: the area under the curve (AUC) was 0.67 (95% CI 0.52–0.82).

A similar approach was used in several other studies that confirmed the presence of statistically significant RLS at rest in patients with and without hemodynamically significant coronary stenosis [15]. Assessment of predictive capacity of RLS in these studies was virtually the same (AUC from 0.72 to 0.75), however, the values of the cutoff varied within a broad range (12.6…18.3%), which precludes consideration of RLS at rest as a predictor for identification of hemodynamically significant coronary stenosis.

Regional longitudinal strain during physical or pharmacological stress may provide a more reliable predictor

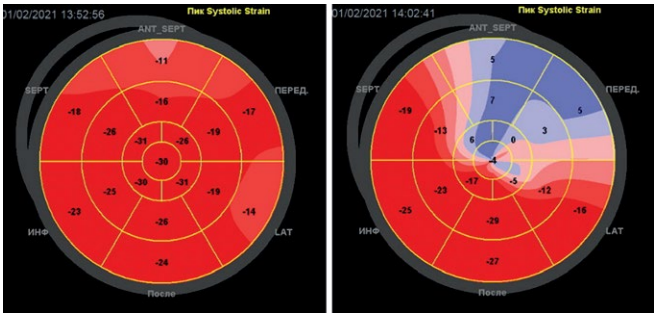


Figure 4. *Left ventricular strain in a patient with hemodynamically significant stenosis of the anterior interventricular branch (LAD) of the left coronary artery before (left) and after (right) dosed physical exercise. Before the exercise, the average longitudinal strain value in the blood supply zone of the LAD is -22%, after the exercise – +2.1%. A positive value of systolic longitudinal strain indicates that the myocardium is stretched during systole, i.e. is in the state of dyskinesis.*

Рисунок 4. *Стрейн левого желудочка у пациента с гемодинамически значимым стенозом передней межжелудочковой ветви (МПЖВ) левой коронарной артерии до (слева) и после (справа) дозированной физической нагрузки. До нагрузки среднее значение продольного стрейна в зоне кровоснабжения ПМЖВ равно -22%, после нагрузки – +2,1%. Положительное значение систолического продольного стрейна свидетельствует, что миокард во время систолы растягивается, то есть находится в дискинезе.*

of hemodynamically significant coronary stenoses than measurements at rest (**Fig. 4**).

Indeed, the study of S.I. Farag *et al*. (2020) [16] showed an almost complete matching of results of assessment of the availability and localization of the hemodynamically significant atherosclerosis on the color map after dobutamine stress testing, and CAD data. The agreement coefficient (kappa) was 0.819, significantly exceeding the kappa coefficient for agreement with visual assessment of RCD during stress echocardiography compared to coronary angiography results (kappa 0.663). The sensitivity of RCD assessment with the color map of the regional longitudinal strain with respect to identification of the hemodynamically significant atherosclerosis was 95%, its specificity was 90%; the respective values of the visual assessment of RCD were 86% and 85%. The results of the study confirm the perspectives of studying the RLS under stress in the diagnostics of transitory myocardial ischemia; however, we found no other papers on this topic in the available literature. Much more frequent were the papers studying the diagnostic capacity of the global longitudinal strain (GLS).

GLS is calculated as the arithmetic mean of the strain values in all segments of the left ventricle. In healthy individuals at rest, the GLS is nearing 20%, and under stress, it increases by approx. 5 percent points. After exercise test on a cycle ergometer, GLS of 67 healthy individuals aged between 23 and 80 increased from the base level of 20.1 ± 1.8% to 25.4 ± 2.0%, p<0.0001 [13]. In 46 patients without CHD but with risk factors of its development (arterial hypertension, Diabetes mellitus, hyperlypidemia, positive family history), the global strain grew to 25 ± 3% (p<0.05) [17]. Administration of dobutamine also leads to GLS growth in healthy individuals less pronounced than under physical stress [18].

GLS reflects contractile capacity of the left ventricular myocardium on the whole, i.e. provides the same information as the ejection fraction; at the same time, it has higher sensitivity and reproducibility in the assessment of the systolic function of the left ventricle [19, 20]. Thus, determination of GLS is recommended for the control of the systolic function of the left ventricle in the use of cardiotoxic drugs, specifically, during chemotherapy of oncological diseases [21, 22], and new diagnostic algorithms to identify heart failure with preserved ejection fraction and diagnostics of the diastolic function of the left ventricle [23–25].

Some studies show that the GLS values at rest differ in patients with and without hemodynamically significant coronary atherosclerosis. A systematic review of these studies showed that the differences in GLS at rest in patients with stenosis (n = 397) and without stenosis (n = 381) are 2 percent points (17.2 ± 2.6 vs. 19.2 ± 2.8%, p <0.0001), AOC varies from 0.68 to 0.80, and the threshold level for the prediction of hemodynamically significant stenosis varies from 17.4 to 19.7%, with sensitivity from 51% to 81% and specificity from 58% to 81% [15]. Based on the obtained data, the authors of the review concluded that GLS at rest has only minor capacity in the prediction of hemodynamically significant stenosis in patients with acute or recurrent chest pains.

A similar conclusion stems from the results of later studies. Thus, in the study of A.I. Stepanova *et al*. (2021) [26], GLS at rest demonstrated no predictive significance with respect to coronary atherosclerosis generally or with respect to severe coronary atherosclerosis (Gensini score ≥35). In the former case, the AOC was 0.52 (95% CI 0.42–0.63, p = 0.59), while in the latter it was 0.63 (CI 0.47–0.73, p = 0.12). However, in some papers, GLS at rest shows very high predictive capacity with respect to hemodynamically significant stenosis of coronary arteries. For example, in the above mentioned study of S.I. Farag *et al.* (2020) [16], the area under curve for GLS at rest was 0.827 (95% CI 0.732–0.921), and in the study of S. Qin et al. [27] it was 0.973.

The studies focusing on prognostic capacity of GLS under physical or pharmacological stress [28–32] demonstrate more uniform results. In the study of A.I. Stepanova et al. (2021) [26], the area under curve for GLS under physical stress as predictor of identification of severe coronary atherosclerosis (Gensini score ≥ 35) was 0.76 (95% CI 0.63–0.89; p <0.001), and the sensitivity and specificity for GLS below 16.9% was 80% and 70%, respectively. In the study of S.I. Farag *et al*. (2020) [16], similar values for global strain under dobutamine stress were 0.837 (95% CI 0.748–0.927), 82.4 and 78.3% for GLS below 12.5%.

In the assessment of results of studies of predictive capacity of GLS with respect to hemodynamically significant coronary atherosclerosis one needs to consider that GLS provides an assessment of the contractive capacity of the left ventricular myocardium on the whole. Its decrease may be related to persistent or transient regional contractility disorders and with a number of other reasons, e.g. type II diabetes mellitus [33, 34], the prevalence of which among patients with severe coronary atherosclerosis is quite high. In the above mentioned study of S.I. Farag *et al*. (2020) [16], 59% of examined patients had the concomitant diabetes mellitus. At the same time, according to C. Philouze *et al*.

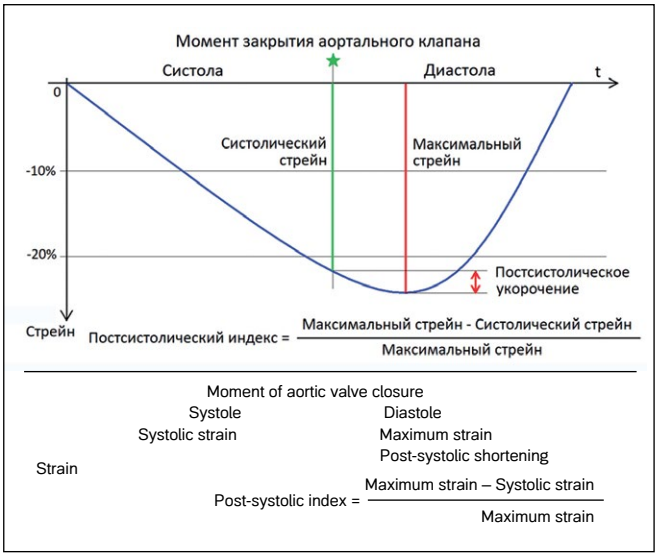


Figure 5. *Schematic representation of the longitudinal strain change graph, in which the maximum longitudinal strain is observed after the completion of left ventricular systole.*

Рисунок 5. *Схематическое изображение графика изменения продольного стрейна, на котором максимальный продольный стрейн отмечается после завершения систолы левого желудочка.*

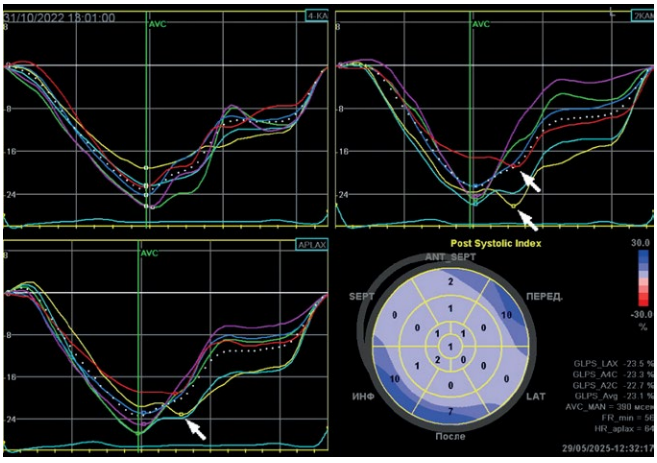


Figure 6. *Results of calculation of the postsystolic index. The graphs of strain changes in 17 segments of the left ventricle during the cardiac cycle are presented. The systolic longitudinal strain corresponds to the point of intersection of the graph with the vertical line AVC (aortic valve closure), reflecting the moment of closure of the aortic valve. Arrows indicate the maximum longitudinal strain recorded after the completion of systole. Segments in which postsystolic shortening is recorded are highlighted in saturated blue on the color map.*

Рисунок 6. *Результаты расчета постсистолического индекса. Представлены графики изменения стрейна в 17 сегментах левого желудочка на протяжении кардиоцикла. Систолическому продольному стрейну соответствует точка пересечения графика с вертикальной прямой AVC (aortic valve closure), отражающей момент закрытия аортального клапана. Стрелки указывают на максимальный продольный стрейн, зарегистрированный после завершения систолы. Сегменты, в которых зарегистрировано постсистолическое укорочение, выделены на цветовой карте насыщенным синим цветом.*

(2018) [35], the increase of GLS under dobutamine stress in patients with diabetes mellitus was much less marked than in healthy individuals. In the control group, GLS under stress increased on average by 3.4 percent points (from 20.8 ± 2.3 to 24.2 ± 2.5%), and in the diabetes group, just by 1 percent point (from 20.2 ± 2.7 to 21.2 ± 2.4%). The authors believe that the mild increase of GLS could be the excessive deposit of epicardial fat in diabetes patients: being a source of proinflammatory and profibrotic cytokines, it adversely affects the contractile capacity of cardiomyocytes [36].

The study of M.J. Mansour *et al*. (2018) [31], the results of stress-echocardiography with speckle tracking were compared not only with CAG data on the severity of coronary atherosclerosis, but also with the presence of concomitant diseases in the patient (cerebrovascular diseases, diabetes mellitus, arterial hypertension), and cardiovascular risk factors (smoking and dyslipidemia). It was found that the lower values of GLS at rest and under stress were identified not only in patients with marked coronary atherosclerosis, but also in patients with multiple concomitant conditions and risk factors which, in the authors' opinion, indicates a subclinical dysfunction of the left ventricle caused by those conditions. Based on the obtained information, the authors conclude that the higher values of GLS allow exclusion of hemodynamically significant stenosis, whereas the lower values of strain provide no foundation for its diagnostics.

As a sensitive marker of disorder of the global systolic function of the left ventricle, the GLS can hardly become an effective means of diagnosing regional contractility disorders. Assessment of longitudinal strain in the perfusion regions under stress seems to be more promising, but, as we mentioned earlier, this problem requires further research. Moreover, the assessment of global and regional longitudinal strain has some limitations related to the image quality and some rhythm disorders (e.g., ventricular bigeminy).

POST-SYSTOLIC SHORTENING

The systole of the left ventricle finishes at the moment of aortic valve closure. By that time, the majority of segments reach the state of maximum contraction and begin elongating. However, some segments continue contracting even after the systole of the ventricle is over. This phenomenon is referred to as 'post-systolic shortening' (PSS). Quantitative values of the PSS is the difference between the maximum and systolic strain (PSS = maximum longitudinal strain – systolic longitudinal strain) and the post-systolic index (PSI), that is the percentage of this difference from the maximum longitudinal strain (PSI% = PSS/ maximum longitudinal strain, **Fig. 5**).

PSI is calculated for each segment of the left ventricle and is represented on the color map (**Fig. 6**). In order to analyze the obtained data, the average PSI value is calculated in all segments of the left ventricle (global PSI) or in the segments referring to perfusion are of some coronary artery or other (regional PSI).

According to P. Brainin *et al*. (2019) [37], in healthy individuals PSS is registered on average in 80% segments of the left ventricle. At the same time, the median PSS is 0.4% [interquartile range 0.2; 0.8%], and the median PSI is 2% [0.7; 4.8%]. The mechanism of physiological PSS is related to the measurement of geometry of the left ventricle at the stage of isovolumic relaxation, i.e. in the period from the closure of aortic valve to the opening of the mitral valve. At this stage, the cavity of the left ventricle turns

from elongated on the longitudinal axis to near spherical in shape. Singe the surface area of a sphere is less than the area of any non-spherical body of the same volume, the area of the myocardium surrounding the cavity of the left ventricle and the distance between its neighboring points decrease. Therefore, the physiological PSS is shortening, not the contraction, of the myocardium.

From the clinical perspective, the PSS phenomenon is interesting because the reason for its onset might be myocardial perfusion disorder. In some experimental studies on animals it was shown that the occlusion of the coronary artery results in the lowering of the longitudinal strain and increase of the PSI in the perfusion area of that artery, and the restoration of the blood supply is accompanied by a quick restoration of the strain and relatively slow return to baseline levels of PSS indices. The phenomenon of delayed recovery of PSS indices after ischemia resolution has been termed "ischemic memory" [8, 38].

The relation of PSS and TMI has been demonstrated in several studies involving stress-echocardiography with dobutamine or exercise stress [38–40]. Thus, the work of A.I. Stepanova et al. (2022) [39] showed, that in the individuals without coronary atherosclerosis (Gensini score = 0), moderate (<0 Gensini score <35) and severe (Gensini score ≥35) coronary atherosclerosis at rest, the global PSI did not have statistically significant difference and was 2.0 [0.9; 4.1], 2.1 [1.3; 4.2] and 2.7 [1.9; 5.2]%, respectively. After a predefined exercise on the treadmill, the global PSI increased to 3.8 [2.2; 6.8], 3.4 [2.2; 6.2] and 8.9 [3.8; 10.7]%, respectively, which resulted in the appearance of statistically significant differences between patients with and without severe coronary atherosclerosis ($p = 0.012$). The area under curve for global PSI as a predictor of severe coronary atherosclerosis was 0.74 (95% CI 0.63–0.85; $p < 0.001$), and the sensitivity and specificity of the criterion 'global PSI > 4.9%' was 75% and 61%.

Similar results were arrived at in the study of E. Rumbinaite et al. (2020 [40], where stress echocardiography under dobutamine stress was performed for 83 patients with pre-test chance of CAD, in 45 of which CAG identified hemodynamically significant stenosis of coronary arteries. Initially, the global PSI in patients with and without stenosis was 4.59 ± 3.04 and 4.07 ± 1.37% ($p = 0.32$), and under dobutamine stress it increased respectively to 10.46 ± 3.42 and 5.23 ± 1.96% ($p = 0.02$). The area under curve for global PSI under stress was 0.724 ($p = 0.04$), the sensitivity and specificity of the criterion 'global PSI > 6.46%' was 70% and 74%.

This study focused not only on the global but on regional PSIs on the perfusion areas of three coronary arteries as well. Initially, in the perfusion area of the anterior intraventricular branch the regional PSI in patients with and without hemodynamically significant stenosis was 6.87 ± 3.32 and 4.65±2.32%, respectively; after dobutamine stress, 11.59 ± 5.21 and 6.43 ± 3.21 ($p = 0.02$). In the perfusion area of the right coronary artery, the respective initial values of PSI were 6.51 ± 3.14 and 4.58 ± 2.42% ($p = 0.34$), under stress: 10.71 ± 4.21 and 5.59 ± 2.46 ($p = 0.03$). In the basin of the circumflex branch of the left coronary artery, the initial PSI values were 6.65 ± 3.14 and 5.01 ± 2.34% ($p = 0.53$), under stress: 7.13 ± 4.16 and 5.78 ± 2.67% ($p = 0.18$). Thus, dobutamine stress results in the increase of both the global PSI and the regional PSI in the perfusion area of the affected artery, yet the initial values of the regional PSI and their increase under stress differ in various vascular areas. Obviously, these differences form additional complications for the interpretation of results of assessment of the regional PSI.

The possibility of using the changes in the PSI under stress to identify individuals with hemodynamically significant coronary atherosclerosis may be considered proven; at the same time, a number of problems remain unanswered.

First, we should mention the lack of a generally recognized criterion of diagnosing the pathological PSS. Quite frequently, PSI value above 20% is used as this criterion, which was suggested to diagnose pathological PSS using the data of Doppler tissue examination. At the same time, we already mentioned that in the patients with hemodynamically significant atherosclerosis the PSI in the perfusion area of the affected artery under stress increases to approx. 10%. The PSI increase in this case is, without doubt, pathological, but it is not anywhere near 20%. Thus, the question of diagnostic criteria of pathological PSS remains unanswered and requires further research.

In the majority of studies focusing on the correlation of PSS with myocardial ischemia, considerably high sensitivity of PSI increase as the sign of myocardial ischemia was demonstrated; however, its specificity requires further research: pathological PSS is identified in the hypertrophic, dilatation and stress cardiomyopathy, arterial hypertension and aortic stenosis [41]. It is evident that in order to use the PSI in clinical practice a criterion for differential diagnosis of ischemia- and non-ischemia-mediated changes of that indicator.

Another question in need of further research is that of the mechanisms of development of pathological PSS, and these include the following: 1) delayed start of contraction of a segment of myocardium due to late arrival of the excitation wave; 2) delayed contraction of the affected (e.g., ischemic) section of the myocardium; 3) deformation of the section of the myocardium that lost its contractile capacity during contraction and relaxation of the myocardium around.

A visual representation of the role of disorders of intraventricular conductivity in the development of PSS is shown in the color maps shown in **Fig. 7** built in the course of intracardial electrical stimulation of various sections of the conductive system. Evidently, intraventricular conduction disturbances virtually preclude the identification of PSS from other origins. It is plausible that local abnormalities in intraventricular conduction contribute to the development of pathological PSS in ischemic myocardial regions, though this hypothesis requires further investigation.

Delayed myocardial contraction is indicated as the cause of PSS in the outcomes of the study of C. Eek *et al.* (2011) [42], where indices of longitudinal strain and PSS in the area of affected coronary artery were compared before and after revascularization in patients with non-ST-segment elevation myocardial infarction. It was found that after revascularization the recovery of the systolic function occurs in those segments in which marked PSS was registered on the background of acute ischemia. This result enabled the authors to conclude that registration of PSS in the segments perfused by the affected coronary arteries shows preserved viability of the ischemic myocardium.

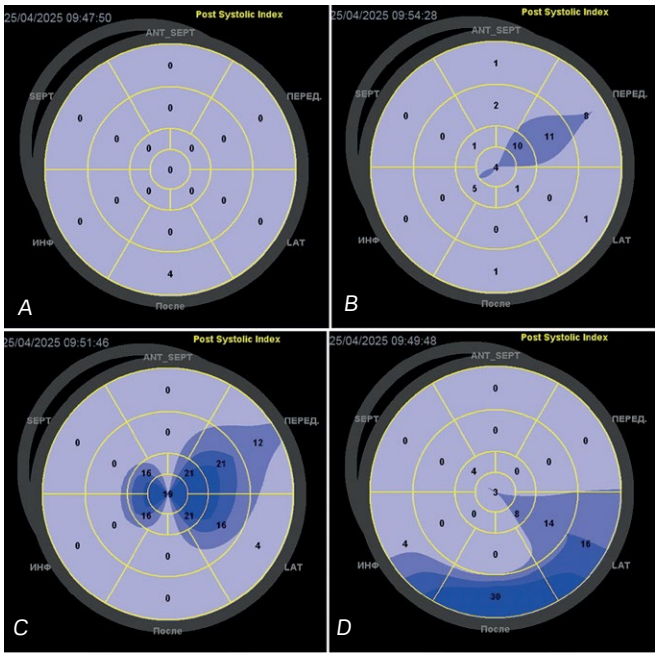


Figure 7. *Effects of the excitation wave propagation path along the left ventricle on the post-systolic contraction. A: sinus rhythm, B: electrical cardiac stimulation in the His bundle region, C: in the region of the middle third of the interventricular septum, D: in the region of the apex of the right ventricle.*

Рисунок 7. *Влияния пути распространения волны возбуждения по левому желудочку на постсистолическое сокращение. А – синусовый ритм, Б – электрокардиостимуляция в области пучка Гиса, В – в области средней трети межжелудочковой перегородки, Г – в области верхушки правого желудочка.*

However, in the similarly designed studies of C. Terkelsen *et al*. (2007) [43] and P. Brainin et al. (2018) [44], dynamic follow-up did not identify recovery of the systolic function of the segments with marked PSS. Moreover, they identified a correlation of PSS with the further development of heart failure, which shows non-viability of the myocardial section with a marked PSS and enables connection of the phenomenon with the deformation of the myocardial section that lost its contractile capacity during relaxation of the muscular tissue around.

The mechanism of the onset of PSS in the myocardium incapable of contraction is easily understood on the example of a myocardial area in the state of dyskinesis. During the systole, this section protrudes beyond the contour of the left ventricle which results in the increase of area of that section and increase of distance between its neighboring points. During the diastole, the protrusion disappears, and the area of the dyskinetic section and the distance between its neighboring points decrease: this is registered as the PSS.

Thus, the question of development of pathologic PSS is directly related to the clinical interpretation of this phenomenon and, beyond doubt, is worthy of further research. This might be confirmed by results of speckle-tracking echocardiography shown in **Fig. 8.** In the patient with 95% stenosis of the circumflex branch of the LCA in the physical stress test, RCD (hypokinesia) in the basal and the medial posterior lateral segments were identified visually. At the same time, the values of the longitudinal systolic strain during the stress increased in all segments including the perfusion area of the affected artery (**Fig. 8 A, C**), which contradicts the results of visual assessment of the stress test and the CAG data. Yet the post-systolic index in the perfusion area of the circumflex branch of the LCA increased from 0 to 27% (**Fig. 8 B, D**), thus confirming the adequacy of visual assessment of the stress test. Based on the available knowledge of mechanisms of development and clinical significance of changes of indices of deformation under physical stress, it does not seem possible to explain the results of this study.

CONCLUSION

Currently, the assessment of left ventricular deformation parameters during physical or pharmacological stress cannot be considered a replacement for the conventionally used visual evaluation of regional contractility dysfunction. At the same time, more knowledge on the mechanisms and clinical significance if carious mechanisms of deformation of the myocardium, as well as development of the very method of speckle-tracking echocardiography and algorithms of automated processing of data thus obtained, forms a realistic perspective of its implementation in clinical practice as the principal method of identification of transitory RCDs in patients with hemodynamically significant coronary atherosclerosis.

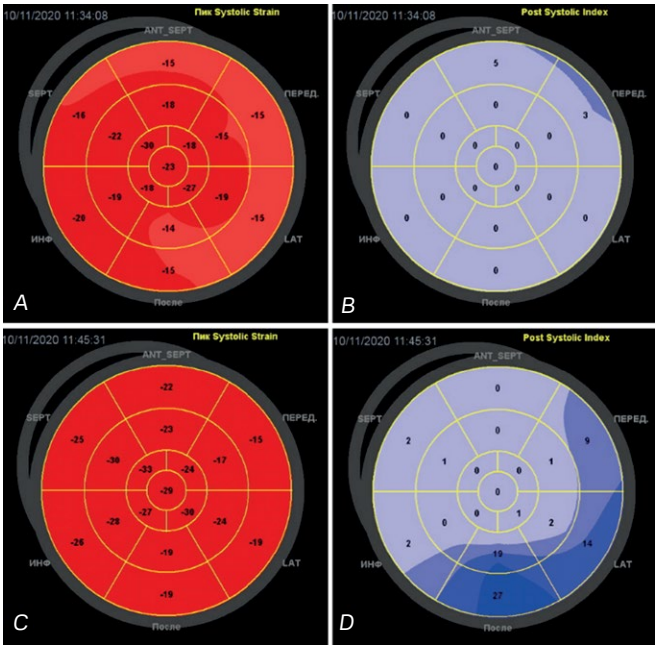


Figure 8. *Results of speckle-tracking echocardiography at rest (A, B) and after dosed physical exercise (C, D) in a patient with 95% stenosis of the circumflex branch of the left coronary artery.*

Рисунок 8. *Результаты speckle-tracking эхокардиографии в покое (А, Б) и после дозированной физической нагрузки (В, Г) у пациента с 95% стенозом огибающей ветви левой коронарной артерии.*

ADDITIONAL INFORMATION	ДОПОЛНИТЕЛЬНАЯ ИНФОРМАЦИЯ
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Conflict of interest. The authors declare that there are no obvious or potential conflicts of interest associated with the content of this article.	***Конфликт интересов.*** Авторы декларируют отсутствие явных и потенциальных конфликтов интересов, связанных с содержанием настоящей статьи.
Compliance with Ethical Standards. The study was approved by the local Ethics Committee of the Tver State Medical University of the Ministry of Health of the Russian Federation, Protocol No.4 dated 16.04.2025).	***Соответствие нормам этики.*** Исследование одобрено локальным этическим комитетом ФГБОУ ВО Тверской ГМУ Минздрава России (протокол №4 от 16.04.2025 г.)
Contribution of individual authors. Nikolaeva T.O.: contribution to obtaining, analyzing literature data and writing of the text of the article. Mazur V.V.: contribution to obtaining results, editing of the text of the article, provision of illustrative material from the author's archive. Mazur E.S.: development of the article concept, data analysis, making significant important edits to the manuscript in order to increase the scientific value of the article. The authors gave their final approval of the manuscript for submission, and agreed to be accountable for all aspects of the work, implying proper study and resolution of issues related to the accuracy or integrity of any part of the work.	***Участие авторов.*** Николаева Т.О. – вклад в получение, анализ данных литературы и написание текста статьи. Мазур В.В. – вклад в получение результатов, редактирование текста статьи, предоставление иллюстративного материала из авторского архива. Мазур Е.С. – разработка концепции статьи, анализ данных, внесение в рукопись существенно важной правки с целью повышения научной ценности статьи. Все авторы одобрили финальную версию статьи перед публикацией, выразили согласие нести ответственность за все аспекты работы, подразумевающую надлежащее изучение и решение вопросов, связанных с точностью или добросовестностью любой части работы.

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