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Global Longitudinal Strain in different forms of exercise-related cardiac remodelling

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Abstract

Background: Cardiac structure and haemodynamic parameters reflect distinct patterns of adaptation to exercise. Global longitudinal strain (GLS) is an echocardiographic measure of myocardial deformation that may detect subtle changes in systolic function. **Aim:** This paper aims to analyze changes in GLS associated with different athletic disciplines, training characteristics and acute exercise, and to discuss the clinical interpretation of GLS in physiological versus pathological cardiac remodelling. **Material and methods:** Literature data on the use of GLS in athletes and in the assessment of exercise-related cardiac remodelling were analyzed. This narrative review included studies published between 2010 and 2025 retrieved from PubMed, Scopus, and Web of Science using combinations of the terms *GLS*, *speckle-tracking echocardiography*, *strain analysis* and *athlete's heart*. **Results:** The reviewed studies demonstrate heterogeneous GLS patterns among athletes and across athletic disciplines. Acute prolonged exercise may produce transient changes in myocardial strain, whereas chronic training is associated with variable adaptations depending on exercise modality and cardiac remodelling. **Conclusions:** GLS should not be interpreted using a single athlete-specific

threshold. Its interpretation should consider athletic discipline, training status, ventricular geometry, loading conditions and the timing of measurement relative to exercise.

Keywords: GLS, speckle-tracking echocardiography, athlete's heart, exercise

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Abbreviations:

GLS – global longitudinal strain
GCS – global circumferential strain
GRS – global radial strain
GSR – global strain rate
GWI – global work index
HCM – hypertrophic cardiomyopathy
HTN – hypertension
LAVI – left atrial volume index
LVEDD – left ventricular end-diastolic diameter
LVEDV – left ventricular end-diastolic volume
LVEF – left ventricular ejection fraction
LVESV – left ventricular end-systolic volume
LVH – left ventricular hypertrophy
LVMI – left ventricular mass index
NT-proBNP – N-terminal pro-B-type natriuretic peptide
RV GLS – right ventricular global longitudinal strain
SBP – systolic blood pressure
STE – speckle-tracking echocardiography
VA coupling – ventricular–arterial coupling
VO₂/kg – oxygen consumption per kilogram of body weight

Introduction

Regular physical activity, particularly competitive sports, induces physiological and structural cardiac adaptations collectively referred to as the athlete's heart [1]. In clinical practice, distinguishing physiological cardiac remodelling from pathological myocardial hypertrophy and other forms of maladaptive remodelling can be challenging, particularly in competitive athletes [2-4]. Conventional echocardiographic parameters, particularly left ventricular ejection fraction (LVEF), may remain within the normal range despite subtle alterations in myocardial function [5-7].

Global longitudinal strain (GLS) is an echocardiographic parameter that quantifies longitudinal myocardial deformation and is commonly assessed using speckle-tracking echocardiography (STE) [5-7]. As a relatively sensitive marker of myocardial deformation, GLS may provide additional information on cardiac adaptation to exercise and help characterize differences between physiological and pathological remodelling. However, GLS values may be influenced by image quality, loading conditions, heart rate, blood pressure, acquisition protocols, and software or vendor-related factors, which should be considered when interpreting measurements.

The cardiac response to exercise is influenced by the type, intensity, and duration of training, as well as by individual characteristics and the degree of chronic adaptation. Consequently, GLS findings may differ between athletic disciplines and may also change following acute, particularly prolonged, exercise. These changes do not necessarily indicate myocardial injury or ischaemia and should be interpreted in the context of the underlying cardiac phenotype and haemodynamic conditions.

This narrative review aims to summarize the available evidence on GLS in athletes, with particular emphasis on differences between athletic disciplines, the effects of chronic training and acute exercise, and its potential role in distinguishing physiological cardiac adaptation from pathological remodelling.

Materials and Methods

This narrative review was conducted to summarize the available evidence on the use of global longitudinal strain (GLS) in athletes and physically active individuals, with particular emphasis on differences between athletic disciplines, chronic training adaptations, acute exercise-induced changes, and the differentiation of physiological and pathological cardiac remodelling.

A literature search was performed in PubMed, Scopus, and Web of Science databases for studies published between January 2010 and December 2025. The search strategy included combinations of the following terms: “global longitudinal strain”, “GLS”, “speckle-tracking echocardiography”, “athlete’s heart”, “athletes”, “sports”, “exercise”, “training”, “cardiac remodelling” and “myocardial strain”. Additional relevant publications were identified by screening the reference lists of selected articles.

Studies were considered relevant if they evaluated GLS or other myocardial deformation parameters in athletes, physically active individuals, or populations relevant to the assessment of exercise-related cardiac remodelling. Studies addressing acute exercise responses, chronic training adaptations, differences between athletic disciplines, and the distinction between physiological and pathological cardiac remodelling were included. Studies unrelated to myocardial deformation or exercise-associated cardiac adaptation were excluded.

The identified literature was reviewed qualitatively. Due to substantial heterogeneity in study populations, athletic disciplines, exercise protocols, echocardiographic techniques, follow-up periods, and reported GLS parameters, the findings were not subjected to quantitative meta-analysis. The selected studies were subsequently organized according to the main topics of the review, including GLS assessment in athletes, sport-specific differences, chronic training adaptations, acute exercise-induced changes, and the differentiation of physiological from pathological cardiac remodelling.

GLS

Global longitudinal strain (GLS) is an echocardiographic parameter that quantifies longitudinal myocardial deformation along the long axis of the left ventricle. It is commonly assessed using speckle-tracking echocardiography (STE), a Doppler-independent technique that tracks acoustic markers (“speckles”) within the myocardium throughout the cardiac cycle [5-7]. Because longitudinal myocardial deformation is largely determined by the subendocardial longitudinal fibres, GLS may be sensitive to subtle changes in myocardial systolic function that can occur before a reduction in left ventricular ejection fraction (LVEF) becomes apparent.

GLS is expressed as a percentage, with negative values reflecting myocardial shortening during systole. More negative values generally indicate greater longitudinal shortening, whereas values closer to zero indicate reduced longitudinal deformation. GLS measurements may be influenced by image quality, loading conditions, heart rate, blood pressure, acquisition protocol, and differences between echocardiographic software and vendors. Therefore, GLS should be interpreted in the appropriate clinical and physiological context rather than according to a single universal threshold.

Table 1 summarizes selected studies evaluating GLS in athletes and different forms of exercise-related cardiac remodelling.

Table 1. Studies interpreting GLS values in athletes

Author (Year)	Study and Control Groups	Number	Key GLS findings	p-value
D'Andrea et al. [8] (2010)	Endurance athletes/ HCM	50 / 35	RV GLS correlated with LVEDD in athletes, septal thickness in HCM, and maximal workload overall	<0.0001
Afonso et al. [9] (2012)	HCM / HTN LVH / athletes with LVH / control	56 / 34 / 27 / 12	GLS/strain patterns differed between physiological and pathological LVH	<0.001
Szauder et al. [10] (2015)	Marathon runners /bodybuilders / control	24 / 14 / 15	GLS was less negative in runners; GCS correlated with SBP and LV mass	<0.05
D'Ascenzi et al. [2] (2015)	Team athletes	91	A slight change in GLS was observed after training	0.044
Jouffroy et al. [11] (2015)	Ultramarathon runners	66	GLS decreased significantly during prolonged exercise; a significant reduction in LV systolic function	0.0008

			was observed in 48% of participants	
Lin et al. [12] (2016)	American football players: linemen / non- linemen	30 / 57	GLS decreased in linemen, whereas it increased in non- linemen; GLS changes in linemen correlated with SBP and LVMI	<0.001
Sengupta et al. [13] (2018)	Amateur marathon runners	50	GLS and GCS became less negative at the post-marathon assessment	0.003
Lakatos et al. [3] (2020)	Athletes/control	138 / 50	GLS correlated with VO ₂ /kg	<0.001
Bhatt et al. [14] (2020)	NBA basketball players	550	GLS was less negative in athletes with LVH	0.02
Zimmermann et al. [4] (2021)	Winter sports athletes	32	GLS varies between disciplines	0.0003
Bewarder et al. [15] (2022)	Athletes / LVH /control	78 / 88 / 37	GLS values differed between physiological and pathological LV hypertrophy	<0.001
Tunca et al. [16] (2025)	Women's rugby players	66	GLS was lower in rugby players; inversely correlated with	≤0.013

GLS as a Marker of Myocardial Adaptation to Exercise

GLS may provide additional information on myocardial functional adaptation to exercise that is not apparent from conventional echocardiographic parameters. Its sensitivity to changes in longitudinal myocardial deformation makes it potentially useful for assessing both chronic training-related remodelling and transient responses to acute exercise. However, changes in GLS are not specific to myocardial ischaemia and may also reflect alterations in preload, afterload, heart rate, blood pressure, and other physiological responses to exercise.

This potential role of GLS in differentiating physiological from pathological cardiac remodelling is supported by the findings of Bewarder et al. [15]. They reported that GLS was significantly reduced in patients with pathological hypertrophy compared with athletes and healthy controls. In individuals with septal wall thickness >11 mm, a GLS threshold of $\geq -18\%$ showed 79% specificity for distinguishing pathological from physiological hypertrophy. However, this threshold should not be interpreted as a universal diagnostic cut-off, as GLS is influenced by loading conditions, acquisition technique, vendor-related factors, and patient characteristics.

GLS in Stress Echocardiography

Stress echocardiography allows myocardial function to be assessed under increased haemodynamic demand and may reveal abnormalities that are not apparent at rest. GLS can provide additional information in this setting, particularly when LVEF remains preserved. Broberg et al. [17] demonstrated that childhood cancer survivors treated with anthracyclines had lower GLS, global strain rate, and LVEF at rest compared with controls, while LVEF remained within the normal range in the study group. During dobutamine stress, changes in GLS and global strain rate were also significantly lower in the study group, whereas the change in LVEF did not differ significantly from controls. Similar findings were reported by Niemela et al. and Armstrong et al. [18,19]. These studies support the ability of GLS to detect subtle changes in myocardial function. However, these studies involved non-athletic populations and therefore provide methodological rather than athlete-specific evidence.

The Effect of Exercise on GLS Values

Szauder et al. [10] conducted an echocardiographic study involving 24 marathon runners, 14 bodybuilders, and 15 healthy volunteers. Compared with the other groups, global circumferential strain (GCS) was reduced in bodybuilders, whereas GLS was less negative in marathon runners, while global radial strain (GRS) did not differ significantly between the groups ($p < 0.05$). Among bodybuilders, GCS was correlated with systolic blood pressure (SBP) and left ventricular (LV) mass ($p < 0.05$ and $p < 0.01$, respectively). These findings suggest that myocardial deformation patterns may differ according to the predominant type of exercise. They also indicate that strain imaging may provide additional information about differences in LV remodelling between endurance and strength athletes that may not be fully captured by conventional echocardiographic parameters.

In the study by Lin et al. [12], changes in GLS were assessed in American football players before and after the competitive season. GLS decreased in linemen ($p < 0.001$), whereas it increased in non-linemen ($p < 0.001$). Among linemen, the decrement in GLS was inversely correlated with changes in systolic blood pressure ($p = 0.009$) and LV mass index ($p = 0.003$), whereas these associations were not observed in players occupying other positions. These findings suggest that the haemodynamic characteristics associated with different playing positions may influence cardiac remodelling and myocardial deformation. In particular, the predominance of isometric exercise among linemen may contribute to a different pattern of LV adaptation compared with players exposed to a greater proportion of isotonic exercise. However, changes in GLS should not be interpreted in isolation as evidence of pathological remodelling, and further assessment of blood pressure, LV geometry, and other clinical parameters is required to determine their significance.

In a multicenter study by Tso et al. [20] involving 200 American football players, ventricular-arterial (VA) uncoupling was associated with higher blood pressure and increased afterload. Higher SBP and less negative GLS were associated with increased EA/ELV ($p = 0.02$ and $p < 0.001$, respectively), suggesting a relationship between increased vascular load, ventricular-arterial interaction, and myocardial deformation. These findings indicate that haemodynamic loading may be an important factor influencing GLS in athletes and should be considered when interpreting strain measurements.

A study by Tunca et al. [16] involving 66 female rugby players demonstrated associations between lower GLS values, higher BMI ($p = 0.013$), and longer duration of sporting activity ($p = 0.001$). These findings suggest that myocardial deformation may be related to both body

composition and training history. However, the cross-sectional nature of such associations does not establish a causal relationship between long-term exercise exposure and myocardial remodelling. Overall, the available evidence suggests that GLS may reflect differences in cardiac adaptation according to the type, duration, and haemodynamic characteristics of physical activity, although the direction and clinical significance of these changes remain heterogeneous.

GLS and Training Time and Volume

The study by D'Ascenzi et al. [2] investigated the effects of training on GLS in 91 elite athletes, including soccer, basketball, and volleyball players. After an 18-week training program, LV mass, left ventricular end-diastolic volume (LVEDV), and left ventricular end-systolic volume (LVESV) increased significantly ($p < 0.0001$, $p = 0.0001$, and $p < 0.0001$, respectively). At the same time, a slight but significant change in GLS was observed ($p = 0.044$). Resting heart rate was the only independent predictor of LV GLS after training ($p = 0.005$). These findings indicate that substantial structural LV remodelling during training was not accompanied by impaired longitudinal myocardial deformation. The slight change in GLS, despite the increase in LV mass and volumes, suggests that longitudinal myocardial deformation was largely preserved during training. Zimmermann et al. [4] assessed cardiac adaptation to exercise in 12 competitive biathletes, 10 cross-country skiers, and 10 ski-mountaineering athletes. LV GLS values differed significantly between the groups, with the least negative values observed in ski-mountaineering athletes ($p = 0.0003$). LVEF, LV mass index, and left atrial volume index (LAVI) also differed significantly between the groups ($p = 0.0001$, $p = 0.0078$, and $p = 0.0052$, respectively). These findings suggest that cardiac structure and myocardial deformation may vary between different types of winter sports. However, the small sample size limits the generalizability of these findings, and larger studies including athletes of both sexes are needed to determine whether these differences are consistently associated with specific training characteristics.

In a study by Lakatos et al. [3], cardiac remodelling was assessed in 138 elite athletes and 50 healthy individuals using three-dimensional echocardiography. LV GLS was significantly associated with oxygen uptake normalized to body mass (VO_2/kg) during treadmill exercise ($p < 0.001$). These findings suggest a relationship between longitudinal myocardial deformation and exercise capacity. The combination of LV GLS, maximal left atrial volume (LAV_{max}), and left atrial passive emptying fraction may therefore provide additional information when assessing functional cardiac adaptation to exercise. The findings also support the potential

relevance of left atrial remodelling as part of the broader assessment of cardiovascular adaptation in athletes.

Acute Exercise and Transient Changes in GLS

The article by Jouffroy et al. [11] summarized echocardiographic findings obtained in 66 long-distance runners before and after an ultramarathon, as well as at the 21st and 53rd kilometers of the race. Two patterns of left ventricular systolic response to prolonged exercise were identified. A significant reduction in LV systolic function was observed in 48% of participants, whereas GLS decreased significantly during prolonged exercise. The authors also suggested that alterations in LV diastolic function may precede changes in systolic function during prolonged exercise. These findings demonstrate substantial interindividual variability in the myocardial response to acute, prolonged exercise.

Sengupta et al. [13] investigated echocardiographic changes in 50 amateur marathon runners by comparing measurements obtained before a 4-month training period and at a post-marathon assessment performed within 10 days of the race. GLS and GCS became less negative after the race ($p=0.003$ and $p=0.001$, respectively), whereas the change in GRS was not significant. NT-proBNP, LVEDV, and LV mass also increased significantly ($p=0.001$, $p<0.001$, and $p<0.001$, respectively), accompanied by an increase in LVEF ($p<0.001$). These findings indicate that prolonged strenuous exercise may be associated with transient changes in myocardial deformation and cardiac loading. However, changes in GLS should not be interpreted as direct evidence of myocardial injury or ischaemia, and their underlying mechanisms and reversibility require further investigation. Potential mechanisms may include alterations in loading conditions, autonomic regulation, myocardial contractility, and metabolic stress associated with prolonged exercise.

Overall, the available evidence suggests that acute prolonged exercise can produce transient changes in GLS, but the direction and magnitude of these changes are heterogeneous between individuals and studies. Therefore, GLS measurements obtained after strenuous exercise should be interpreted in the context of exercise duration and intensity, haemodynamic conditions, and the athlete's baseline cardiac phenotype.

Limitations

Several limitations should be considered when interpreting GLS in athletes. First, there is currently limited availability of standardized reference values specifically for athletic populations [15,23]. GLS values may vary according to age, sex, training status, athletic discipline, and cardiac phenotype. Consequently, the use of a single universal GLS threshold for distinguishing physiological adaptation from pathological remodelling in athletes may be inappropriate.

Second, GLS is influenced by several physiological and technical factors. Changes in heart rate following exercise may affect myocardial deformation measurements and complicate the interpretation of post-exercise GLS. Similarly, blood pressure, preload, afterload, and autonomic changes associated with exercise may alter GLS independently of intrinsic myocardial function. Therefore, measurements obtained immediately after strenuous exercise should be interpreted with particular caution.

Body position is another relevant factor. Kandels et al. [21] demonstrated that GLS and global work index (GWI) were significantly lower in the standing position than in the left lateral position ($p < 0.001$ for both), whereas LVEF did not differ significantly between the two positions. The reduction in GLS in the standing position predominantly involved the inferior and posterolateral LV segments. These findings indicate that changes in body position may affect myocardial deformation measurements, most likely through physiological changes in cardiac geometry and preload. Similar positional differences in LV motion, particularly in the mid-basal inferior and posterolateral segments, were reported by Sasaki et al. [22] in healthy volunteers.

In addition, GLS measurements may be affected by image quality, acquisition conditions, and differences between echocardiographic vendors and software platforms. The studies included in this review also differed substantially with regard to athletic discipline, exercise intensity and duration, timing of echocardiographic assessment, participant characteristics, and follow-up periods. Many studies were based on relatively small cohorts, and relatively few provided longitudinal data. These factors limit direct comparison between studies and make it difficult to establish universal patterns of GLS adaptation across different athletic populations.

Finally, although changes in GLS have been observed following both chronic training and acute strenuous exercise, GLS is not a specific marker of myocardial ischaemia or injury. Changes in GLS should therefore be interpreted together with conventional echocardiographic parameters,

LV geometry, haemodynamic conditions, exercise characteristics, and the overall clinical context.

Summary

The evidence summarized in this review indicates that global longitudinal strain (GLS) provides additional information about myocardial deformation in athletes beyond conventional echocardiographic parameters such as left ventricular ejection fraction (LVEF). GLS values may differ between athletic disciplines and may be influenced by the type, intensity, and duration of exercise, as well as by training status and haemodynamic loading conditions. Importantly, differences in GLS do not necessarily indicate pathological myocardial dysfunction and should be interpreted in conjunction with LV geometry, blood pressure, heart rate, and other echocardiographic parameters.

Studies assessing the effects of chronic training demonstrate that physiological cardiac remodelling may occur without deterioration of longitudinal myocardial deformation. In some populations, GLS may remain stable or show favourable changes despite increases in LV mass and ventricular volumes, whereas other studies have demonstrated differences in GLS between specific athletic disciplines. Associations between GLS and exercise capacity, blood pressure, LV mass, and other cardiovascular parameters further suggest that myocardial deformation reflects the complex interaction between cardiac structure, haemodynamic load, and physical performance.

Acute prolonged exercise may also produce transient changes in GLS. However, the direction and magnitude of these changes are heterogeneous between individuals and studies. Therefore, a change in GLS following strenuous exercise should not be interpreted in isolation as evidence of myocardial ischaemia, injury, or pathological remodelling. The interpretation of post-exercise GLS requires consideration of exercise intensity and duration, loading conditions, heart rate, and the athlete's baseline cardiac phenotype.

GLS may also be useful in differentiating physiological athlete's heart from pathological forms of LV hypertrophy, particularly when interpreted together with ventricular geometry and other echocardiographic parameters. Nevertheless, the current evidence is limited by small study populations, heterogeneous athletic disciplines and exercise protocols, differences in measurement conditions and echocardiographic systems, and the lack of standardized athlete-specific reference values.

Overall, GLS appears to be a valuable complementary tool for the assessment of cardiac adaptation to exercise. Its interpretation should remain contextual rather than based on a single universal threshold. Further prospective and longitudinal studies involving larger and more diverse athletic populations, standardized GLS acquisition protocols, and consistent follow-up are needed to establish sport-specific reference ranges and to clarify the clinical value of GLS in distinguishing physiological adaptation from maladaptive cardiac remodelling.

Disclosure

The authors declare that they have no relevant financial or non-financial interests to disclose.

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