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Review

Echocardiographic Ventricular Strain Imaging Reflects Alterations in Myocardial Mechanics During and After Cardiac Surgery

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Abstract

Assessment of ventricular function during and post cardiac surgery remains challenging, as conventional echocardiographic indices may not adequately capture subtle and dynamic changes in myocardial performance. Speckle-tracking echocardiography, particularly left ventricular global longitudinal strain (LV GLS) and right ventricular longitudinal strain, provides a more sensitive assessment of myocardial deformation and has increasingly been applied in the perioperative setting. Available studies indicate that LV GLS may deteriorate following cardiopulmonary bypass (CPB) and cardiac surgical intervention despite relatively preserved left ventricular ejection fraction, highlighting the ability of deformation imaging to detect myocardial dysfunction that may remain undetected by conventional parameters. Right ventricular longitudinal mechanics may also undergo substantial perioperative alterations, reflecting the particular susceptibility of the right ventricle to changes in loading conditions, pulmonary vascular resistance, and ventricular interdependence. Importantly, perioperative strain measurements should be interpreted in the context of the cardiovascular effects of general anesthesia and mechanical ventilation, that may alter preload, afterload, and myocardial contractility. The potential influence of anesthetic technique and depth on myocardial deformation represents an emerging research question requiring further investigation. This narrative review synthesizes current evidence on alterations in left and right ventricular strain and cardiac mechanics in patients undergoing cardiac surgery, with particular emphasis on perioperative changes observed in CABG and valve surgery and on the effects of general anesthesia and mechanical ventilation. By integrating findings across these perioperative stages, it aims to highlight the pathophysiological and procedural factors that should be considered when interpreting myocardial deformation, including anesthesia, altered loading conditions, CPB, and the surgical intervention itself.

Keywords: strain imaging; ventricular mechanics; myocardial deformation; cardiac anaesthesia; CPB; cardiac surgery

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1. Introduction

Perioperative echocardiographic assessment of myocardial function is an essential component of contemporary cardiac surgery and cardiac anesthesia, though challenging due to the extreme volumetric and hemodynamic alterations that may occur [1–3].

Left ventricular (LV) systolic function is mainly assessed by LV ejection fraction (LVEF); however, LVEF is merely a volumetric evaluation of the LV influenced excessively by the loading conditions and the filling pressures and may remain preserved despite subtle abnormalities in endomyocardial performance [2,4,5]. Alternatively, speckle-tracking echocardiography (STE) provides a more detailed assessment of myocardial deformation, with left ventricular global longitudinal strain (LV GLS) being the most commonly used strain parameter for the evaluation of LV systolic mechanics [2], detecting endomyocardial functional abnormalities that are not apparent from conventional indices such as LVEF, although myocardial deformation itself remains influenced by loading conditions [6–8]. Assessment of right ventricular (RV) function is additionally complicated by its complex geometry and contraction pattern [9,10]. Conventional parameters such as tricuspid annular plane systolic excursion (TAPSE) and systolic tricuspid annular velocity (RV S′) primarily reflect annular longitudinal motion and may not fully represent global RV systolic performance [9,11]. RV longitudinal and free-wall strain (RV FWS) may therefore provide complementary information, particularly in the perioperative setting, where major changes in loading conditions, pulmonary vascular resistance (PVR), and ventricular interaction may occur [2,11].

Following coronary artery bypass grafting (CABG), LV GLS may deteriorate despite relatively preserved LVEF, while RV longitudinal mechanics may also undergo substantial postoperative alterations [1,11–15]. In patients undergoing valve surgery, interpretation is further complicated by the distinct loading conditions imposed by the underlying valvular lesion and by their abrupt modification after valve correction, with accompanying alterations in ventricular mechanics [16–19]. Moreover, general anesthesia and intermittent positive-pressure ventilation (IPPV) alter myocardial contractility, systemic vascular resistance, venous return, intrathoracic pressure, and ventricular loading, and may therefore modify both LV and RV deformation even before the onset of surgery [8,20,21]. Consequently, perioperative changes in myocardial deformation may reflect the combined influence of preload-afterload alterations, ventricular interdependence, cardiopulmonary bypass (CPB), mechanical ventilation and the surgical intervention itself [17–20].

This narrative review summarizes current evidence on perioperative changes in LV and RV myocardial deformation following CABG and valve surgery and examines the perioperative factors that should be considered when interpreting cardiac mechanics assessed by ventricular strain, including general anesthesia, mechanical ventilation, loading conditions, CPB, and the surgical intervention itself. In contrast to previous reviews of myocardial strain in perioperative cardiovascular assessment, the present review focuses specifically on the temporal changes in LV and RV mechanics during and after cardiac surgery [22]. By integrating findings from CABG and valve surgery, it aims to study the procedure-related temporal changes in left and right ventricular strain due to induction to anesthesia, mechanical ventilation, and altered loading conditions during CPB and supporting medications.

2. Literature Search Strategy

This narrative review was based on electronic literature searches conducted in PubMed/MEDLINE during August and September 2026. Two complementary search strategies were used. The first combined (“global longitudinal strain” OR “myocardial strain” OR “longitudinal strain” OR “myocardial deformation” OR “speckle tracking”) AND (“cardiac surgery” OR “cardiopulmonary bypass” OR “CABG” OR “coronary artery

bypass grafting" OR "valve surgery"). The second combined ("global longitudinal strain" OR "myocardial strain" OR "longitudinal strain" OR "myocardial deformation" OR "speckle tracking") AND ("general anesthesia" OR "general anaesthesia" OR "anesthesia induction" OR "mechanical ventilation" OR "positive pressure ventilation" OR "PEEP" OR "propofol" OR "sevoflurane"). Relevant references from selected articles and contemporary reviews were also screened manually.

Priority was given to original studies evaluating perioperative LV or RV myocardial deformation in patients undergoing CABG or valve surgery, as well as studies examining the effects of general anesthesia, mechanical ventilation, and altered loading conditions on myocardial strain. Particular consideration was given to prospective studies and larger cohorts, while smaller exploratory or pilot studies were included when they provided relevant mechanistic or perioperative information. Representative studies were selected to cover the main clinical and physiological settings addressed in this review. No formal predefined inclusion or exclusion criteria, systematic study-selection workflow, or risk-of-bias assessment were applied; studies were selected according to their relevance to the aims of this narrative review. The review was limited to echocardiography-based myocardial strain assessment; studies using cardiac magnetic resonance-based feature tracking were considered outside its scope.

3. Coronary Artery Bypass Grafting and Left Ventricular Global Longitudinal Strain

Assessment of LV systolic function in patients undergoing CABG is traditionally based on LVEF, that may remain preserved despite underlying subtle abnormalities in myocardial contractility, due to coronary artery disease. The subendocardial longitudinal fibres are particularly susceptible to ischemic injury that is detected by speckle-tracking LV GLS. Accordingly, a prospective study by Gozdzik et al. showed that LVEF was preserved in approximately 87% of 69 patients scheduled for elective CABG, whereas preoperative GLS was reduced in almost 74% of the patients [12]. Intraoperatively, Chen et al. provided a detailed assessment of the deterioration of longitudinal myocardial mechanics during or shortly after surgical revascularization, using three-dimensional speckle-tracking echocardiography; [23] LV GLS progressively worsened from $-18.6 \pm 4.3\%$ after induction of anesthesia and before sternotomy, to $-17.5 \pm 2.8\%$ before grafting, $-16.2 \pm 3.0\%$ after grafting, and $-16.0 \pm 4.0\%$ after completion of surgery. The reduction became significant after grafting and persisted after chest closure, whereas 3D LVEF, global circumferential strain, LV twist, and torsion remained relatively unchanged throughout the procedure.

A similar discordance between LV GLS and conventional indices was observed by Labus et al., who included 40 patients undergoing uncomplicated, isolated, on-pump CABG [1]. In this prospective study, LV GLS deteriorated significantly from the preoperative assessment to the postoperative examination (-15.2 [IQR, -18.05 to -13.08]% vs. -11.3 [IQR, -15.8 to -9.78]%; $p < 0.001$), whereas LVEF and LV S′ remained essentially unchanged. These findings reinforce the greater sensitivity of longitudinal deformation for detecting subtle postoperative LV dysfunction, while also suggesting that the observed deterioration cannot necessarily be explained by anesthetic induction or sternotomy alone. Consistent with these observations, Howard-Quijano et al. demonstrated significant early postoperative deterioration in both two- and three-dimensional LV GLS in a subgroup of 50 patients undergoing CABG, assessed two to four days after surgery [7]. Two-dimensional LV GLS deteriorated from $-16 \pm 4\%$ to $-12 \pm 4\%$, while 3D GLS from $-12 \pm 4\%$ to $-9 \pm 4\%$ (both $p < 0.005$), whereas neither 2D nor 3D LVEF changed significantly.

The acute strain response following revascularization, however, is not uniform across patients or surgical strategies. A small prospective study by Rong et al. compared intraoper-

ative myocardial deformation before and after on-pump CABG in patients receiving single arterial grafting (SAG) or multiple arterial grafting (MAG) [24]. They found that GLS improved after revascularization in the SAG group but deteriorated in patients undergoing MAG, accompanied by parallel changes in regional longitudinal strain. Of note, patients undergoing MAG also had longer CPB and aortic cross-clamp times and more frequently required postoperative vasopressor and inotropic support.

Importantly, abnormalities in longitudinal deformation may persist beyond the immediate post-bypass period. In a recent prospective study of 51 low-risk patients with preserved LVEF and an uncomplicated course after on-pump CABG, Srivastava et al. demonstrated that LV GLS deteriorated further between the immediate postoperative assessment $-12.8 \pm 3.8\%$ and hospital discharge $-10.2 \pm 2.4\%$ at postoperative day 7–10 ($p < 0.001$) [25]. However, LVEF remained above 50% at both the immediate postoperative and pre-discharge assessments, and the difference in LVEF between these two time points was not clinically significant.

Longer-term studies indicate that this early post-CABG impairment does not necessarily represent permanent myocardial dysfunction. Juhl-Olsen et al. reported a marked early deterioration in GLS after on-pump cardiac surgery, from $-14.5 \pm 3.3\%$ preoperatively to $-10.0 \pm 3.1\%$ on the first postoperative day, with GLS remaining significantly impaired at 30 days ($p = 0.012$) before returning toward baseline by six months, while LVEF remained relatively stable postoperatively [13]. Notably, postoperative GLS was not correlated with peak troponin T levels ($p = 0.57$), suggesting that early impairment in longitudinal deformation is not attributed merely to myocardial injury. Yin et al. studied 145 patients with preserved LVEF before CABG and at 1, 3, and 6 months after surgery [14]. Longitudinal strain showed a mild, non-significant deterioration at one month after surgery, followed by significant improvement relative to baseline at three and six months ($p < 0.05$). In contrast, LVEF, LV dimensions, and stroke volume did not change significantly throughout follow-up. This delayed improvement in longitudinal deformation was attributed to the time required for functional recovery of previously ischemic regions following successful revascularization, while perioperative ischemia–reperfusion injury, CPB, mechanical ventilation, myocardial edema, and surgical myocardial trauma may contribute to delayed recovery during the early postoperative phase. Accordingly, Gozdzik et al. reported reduced GLS during the first three months after CABG, followed by a tendency toward partial recovery and stabilization during longer-term follow-up [12]. An illustrative case example of serial LV GLS changes in a single patient undergoing CABG, from the preoperative assessment through induction of anesthesia and CPB to mid-term postoperative follow-up, is presented in Figure 1. The figure is intended to illustrate within-patient temporal changes and not to represent a generalizable postoperative trajectory. The patient was selected because complete serial echocardiographic examinations of adequate image quality were available across all perioperative and follow-up time points.

Beyond characterizing the temporal evolution of LV longitudinal mechanics after CABG, preoperative GLS may also provide incremental prognostic information. In a cohort of 425 patients undergoing cardiac surgery, including 155 patients undergoing isolated CABG, Ternacle et al. reported 36 early postoperative deaths (8.5%) [6]. In multivariable models adjusted for CPB duration and European System for Cardiac Operative Risk Evaluation I or II, each 1% worsening in preoperative GLS was independently associated with early postoperative mortality (OR 1.10, 95% CI 1.01–1.21; $p = 0.03$) and prolonged inotropic support >48 h (OR 1.15, 95% CI 1.08–1.21; $p < 0.001$) [6].

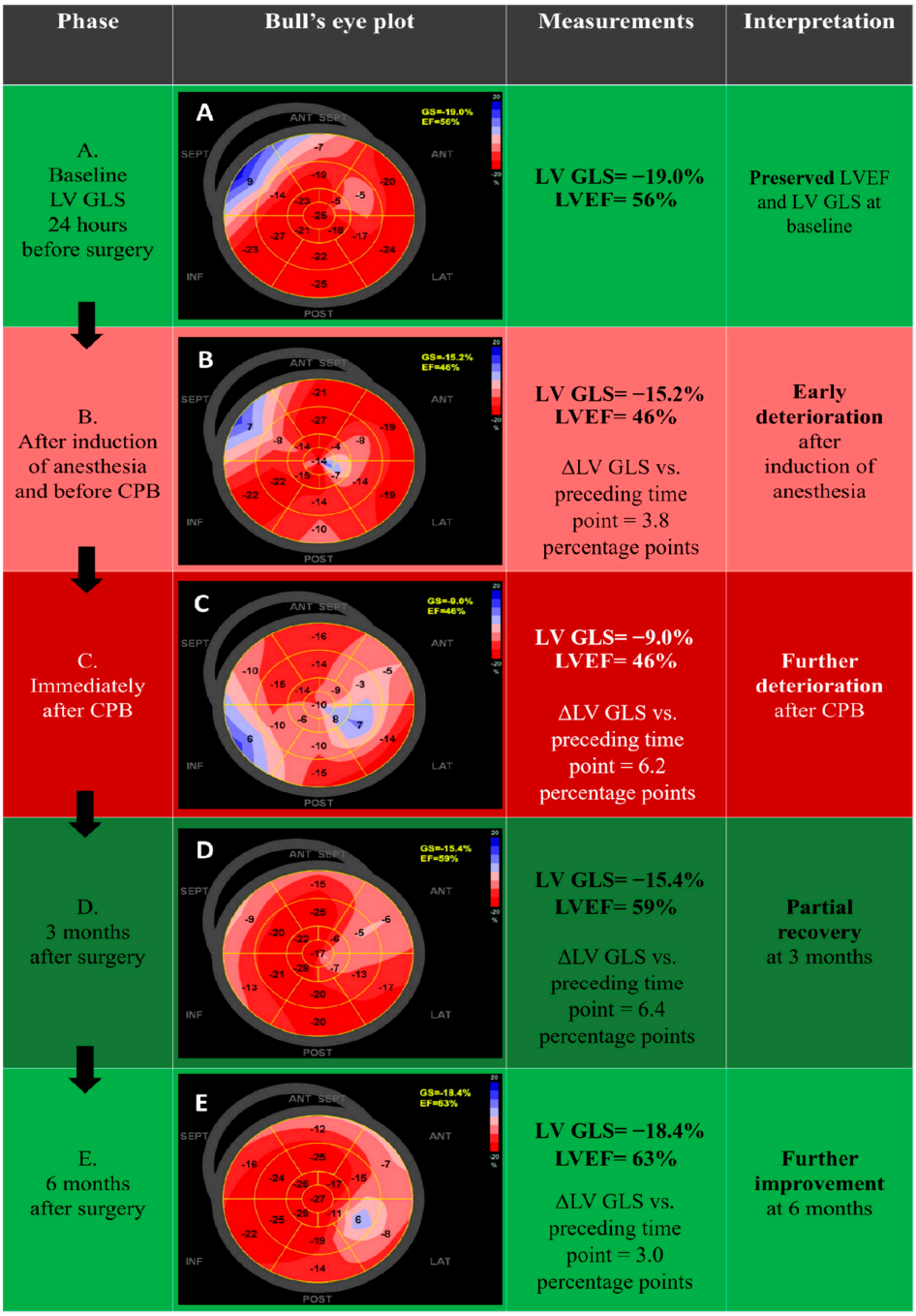


Figure 1. Illustrative case example of serial left ventricular global longitudinal strain (LV GLS) assessment in a single patient undergoing double coronary artery bypass grafting with concomitant surgery for an ascending aortic aneurysm. Measurements were obtained 24 h before surgery (**A**), after induction of general anesthesia and before cardiopulmonary bypass (CPB) (**B**), immediately after CPB (**C**), and at 3 months (**D**) and 6 months (**E**) after surgery. LV GLS and left ventricular ejection fraction (LVEF) values are displayed for each time point. All echocardiographic examinations were performed by transthoracic echocardiography using a Vivid T8 ultrasound system, with strain analysis performed using EchoPAC software version 112.0.1 (GE Healthcare, Chicago, IL, USA). At the post-induction, pre-CPB assessment, blood pressure was 118/45 mmHg, pulmonary capillary wedge pressure was 13 mmHg, mean pulmonary artery pressure was 22 mmHg, right atrial pressure was 17 mmHg, mixed venous oxygen saturation was 83%, cardiac output was 5.4 L/min, and cardiac index was 2.3 L/min/m^2. The patient is presented as an illustrative example of within-patient temporal changes and should not be considered representative of a generalizable postoperative strain trajectory. Changes in LV GLS shown in the figure are calculated relative to the immediately preceding time point.

Taken together, the available evidence suggests that LV longitudinal mechanics after CABG follow a heterogeneous and time-dependent course, characterized by frequent early deterioration despite relatively preserved LVEF, followed by gradual improvement during subsequent follow-up. These perioperative changes should not be interpreted as a direct surrogate of irreversible myocardial injury, since CPB, ischemia–reperfusion, myocardial stunning and edema, loading conditions, vasoactive support, and the timing and modality of echocardiographic assessment may all influence GLS. Nevertheless, reduced preoperative GLS and persistent impairment may provide clinically relevant information beyond conventional measures of LV systolic function.

4. Coronary Artery Bypass Grafting and Right Ventricular Longitudinal Strain

Assessment of RV function after CABG is particularly challenging. The complex geometry of the RV limits the ability of conventional echocardiographic parameters to characterize global systolic performance, while cardiac surgery itself induces substantial changes in RV contraction pattern [10]. In particular, postoperative reduction in longitudinal shortening is well recognized, but may not necessarily indicate a proportional deterioration in global RV pump function [11]. Speckle-tracking-derived indices of RV deformation may therefore provide a more detailed characterization of these perioperative mechanical changes.

Prospective studies in patients undergoing uncomplicated on-pump CABG demonstrate that RV longitudinal deformation may deteriorate substantially during the perioperative period. Labus et al. evaluated 40 patients with preserved preoperative LV and RV function using three-dimensional-derived RV FWS at serial perioperative time points [26]. They revealed that RV FWS was similar between the preoperative awake assessment and the post-induction intraoperative examination and remained unchanged after sternotomy. This finding differs from the modest deterioration in RV longitudinal deformation reported after induction of general anesthesia and positive-pressure ventilation by Dalla et al. [21], highlighting heterogeneity related to patient population, loading conditions, imaging methodology, and timing of assessment. In contrast, a significant deterioration was observed following CABG, with median 3D-RV FWS decreasing from −22.35% preoperatively to −18.5% before hospital discharge ($p = 0.004$). The authors showed that this reduction was accompanied by decreases in TAPSE, RV S′, and 3D-RVEF, whereas RV fractional area change (RV FAC) remained unchanged. Importantly, they state that all measurements were obtained under predefined stable hemodynamic conditions and without vasoactive support, and the postoperative course was uncomplicated. These findings suggest that impairment of RV longitudinal mechanics may reflect a perioperative response to on-pump CABG in otherwise uncomplicated patients, although confirmation in larger cohorts is required.

More detailed insight into the postoperative alteration of RV contraction pattern was provided by Donauer et al.; in their prospective study of 30 patients with preserved biventricular systolic function undergoing on-pump CABG with cardioplegic cardiac arrest, RV GLS was assessed across multiple regions [15]. At the end of the surgery, they observed that peak longitudinal systolic strain decreased markedly in the RV lateral and inferior free wall, the RV outflow tract, and the interventricular septum, with relative reductions ranging from approximately 23% to 41%. The authors reported that the impairment persisted during early postoperative follow-up, with lateral free-wall longitudinal strain decreasing from approximately 22% preoperatively, to 10% four to five days postoperatively ($p < 0.001$), while RVEF remained unchanged. Donauer et al. noted that pericardiotomy itself did not produce a significant immediate reduction in RV longitudinal deformation, suggesting that pericardial incision alone is unlikely to explain the postoperative changes [15]. Instead, CPB, cardio-

plegic arrest, myocardial ischemia or stunning, changes in septal mechanics, and altered loading conditions may all contribute to the postoperative loss of longitudinal shortening.

However, perioperative changes in RV strain after CABG appear to be heterogeneous across studies. Grønlykke et al. prospectively studied 30 low-risk patients undergoing elective on-pump CABG using simultaneous transesophageal echocardiography and pulmonary artery catheter measurements at baseline, after sternotomy, after CPB, and on arrival in the intensive care unit [11]. The authors observed that, immediately after CPB, TAPSE decreased by 43% and S′ by 22%, despite preserved stroke volume and maintained global RV output. In contrast, RV GLS, RVEF, and RV fractional area change remained statistically unchanged throughout the observation period. Moreover, RV GLS changed from −19.9% at baseline to −19.0% after CPB and to −16.6% in the ICU, but the overall change was not statistically significant. These findings further illustrate the dissociation between postoperative longitudinal shortening and global RV performance.

Available evidence suggests that CABG may be associated with substantial alterations in RV mechanics, characterized predominantly by loss of longitudinal contraction rather than uniform deterioration of global RV systolic performance. Longitudinal indices, including RV FWS, may therefore become substantially impaired despite maintained cardiac output or preserved RVEF. These findings suggest that global RV function may remain preserved despite reduced longitudinal shortening; however, a compensatory increase in transverse or circumferential contraction has not been consistently demonstrated and should therefore be considered a proposed mechanism rather than an established finding. Moreover, multidirectional assessment may provide additional insight into perioperative RV mechanics, as longitudinal and circumferential strain components may exhibit different responses to cardiac surgery [27]. Consequently, reduced postoperative RV longitudinal strain should not automatically be interpreted as overt RV failure but rather as a potential manifestation of altered postoperative RV contraction patterns. Differences in strain methodology, imaging modality, timing of assessment, myocardial protection, loading conditions, and vasoactive therapy should also be considered when interpreting the variability observed across studies. Selected studies evaluating perioperative and longitudinal changes in LV and RV longitudinal strain after CABG are summarized in Table 1.

Table 1. Studies evaluating perioperative and longitudinal changes in left and right ventricular global longitudinal strain (GLS) after coronary artery bypass grafting.

Study/Year	Population/ Procedure (n)	Study Design/Key Limitations	Strain Assessment & Software/Vendor	Main Findings
A. Left ventricular strain				
Juhl-Olsen et al. *, 2012 [13]	Mixed on-pump cardiac surgery (n = 39): CABG n = 11; CABG + AVR n = 12; AVR n = 16	Longitudinal single-centre observational study; small mixed surgical cohort; no control group	2D LV GLS; TTE; EchoPAC, GE Healthcare (Vivid Q)	Marked early deterioration, persistent impairment at 30 days, with recovery toward baseline by 6 months
Yin et al., 2013 [14]	Isolated CABG with preserved LVEF (n = 145)	Longitudinal single-centre observational study; selected preserved-LVEF cohort; no control group	2D LV GLS; TTE; QLAB 7.0/Advanced Tissue Motion Quantification, Philips Healthcare (iU22)	Mild early deterioration followed by significant improvement at 3 and 6 months
Howard-Quijano et al. *, 2017 [7]	Mixed cardiac surgery (n = 163); CABG subgroup (n = 50)	Prospective single-centre observational study; heterogeneous surgical cohort; limited subgroup power	2D/3D LV strain; TTE; EchoPAC v110.1.1/4D Auto LVQ, GE Healthcare	Both 2D and 3D LV GLS deteriorated postoperatively despite unchanged LVEF

Table 1. *Cont.*

Study/Year	Population/ Procedure (n)	Study Design/Key Limitations	Strain Assessment & Software/Vendor	Main Findings
Gozdzik et al., 2019 [12]	Elective CABG ($n = 69$)	Prospective, single-centre observational study; modest sample size; attrition during follow-up	2D LV GLS; TTE; EchoPAC, GE Healthcare (Vivid E9)	GLS was frequently reduced preoperatively; early deterioration was followed by partial recovery despite largely preserved LVEF
Rong et al., 2020 [24]	On-pump CABG; SAG $n = 10$ vs. MAG $n = 10$	Prospective exploratory observational study; very small sample; baseline/procedural group differences	2D LV GLS; TEE; QLAB v10.8.5, Philips Healthcare (iE33/EPIQ)	GLS improved after single arterial grafting but deteriorated after multiple arterial grafting
Chen et al., 2021 [23]	CABG ($n = 35$)	Retrospective perioperative observational study; small sample; incomplete 3D datasets; uncontrolled loading conditions	3D LV GLS; TEE; 4D LV Analysis/Image-Arena, TomTec Imaging Systems (Philips Epic 7c/iE33 acquisition)	GLS progressively deteriorated during surgery, mainly after grafting, whereas LVEF remained unchanged
Labus et al., 2022 [1]	Uncomplicated on-pump CABG ($n = 40$)	Prospective single-centre observational pilot study; small highly selected cohort; short follow-up	2D LV GLS; TTE/TEE; 2D Cardiac Performance Analysis, TomTec (Philips EPIQ 7 acquisition)	GLS deteriorated markedly over the perioperative period, while LVEF remained unchanged
Srivastava et al., 2025 [25]	Low-risk on-pump CABG ($n = 51$)	Prospective observational study; highly selected low-risk cohort; TEE–TTE modality change; short follow-up	2D LV GLS; TEE/TTE; EchoPAC 113/AFI, GE Healthcare (Vivid E9)	GLS deteriorated further by postoperative day 7–10, whereas LVEF remained >50%
B. Right ventricular strain				
Grønlykke et al., 2019 [11]	Elective on-pump CABG ($n = 30$)	Prospective single-centre observational study; small highly selected low-risk cohort	RV GLS; serial TEE; 2D Cardiac Performance Analysis, TomTec	RV GLS remained statistically unchanged despite marked reductions in TAPSE and RV S′
Donauer et al., 2020 [15]	On-pump CABG ($n = 30$)	Prospective single-centre observational cohort; small selected population; TTE–TEE comparability limitations	Regional RV longitudinal strain; TTE/TEE; EchoPAC PC 201, GE Healthcare	Postoperative reduction in RV GLS despite preserved global RV systolic performance
Labus et al., 2021 [26]	Uncomplicated on-pump CABG ($n = 40$)	Prospective single-centre observational pilot study; small highly selected cohort; short follow-up	3D RV free-wall strain; TTE/TEE; 4D RV-Function 2.0, TomTec (Philips EPIQ 7 acquisition)	RV free-wall strain remained unchanged after induction and sternotomy but deteriorated significantly before discharge

* Mixed cardiac-surgery cohort; CABG patients constituted a subgroup. Abbreviations: AFI: automated function imaging; AVR: aortic valve replacement; CABG: coronary artery bypass grafting; FWS: free-wall strain; GLS: global longitudinal strain; ICU: intensive care unit; LV: left ventricle; LVEF: left ventricular ejection fraction; MAG: multiple arterial grafting; POD: postoperative day; RV: right ventricle; RVEF: right ventricular ejection fraction; RV S′: systolic tricuspid annular velocity; SAG: single arterial grafting; TAPSE: tricuspid annular plane systolic excursion; TEE: transesophageal echocardiography; TTE: transthoracic echocardiography; 2D: two-dimensional; 3D: three-dimensional.

5. Valve Surgery and Left Ventricular Global Longitudinal Strain

Assessment of left ventricular systolic function in patients undergoing valve surgery is strongly influenced by the loading conditions associated with the underlying valvular lesion. Pressure overload in aortic stenosis (AS), volume overload in aortic regurgitation (AR), and the altered loading conditions associated with mitral regurgitation (MR) affect conventional

indices of systolic function and myocardial deformation differently [16,17]. Accordingly, correction of AS primarily reduces LV pressure afterload, whereas correction of AR or MR reduces volume loading; in MR, elimination of the regurgitant pathway may also acutely increase effective LV afterload and contribute to early GLS deterioration [16,17]. Therefore, changes in LV GLS after valve surgery reflect both endomyocardial function and the altered loading conditions after valve repair or replacement that relieves the hemodynamic burden of the ventricle.

In patients with severe AS, recovery of longitudinal mechanics appears to occur progressively rather than immediately after aortic valve replacement (AVR). Rost et al. analyzed 33 patients before AVR, within the first postoperative week, and at six months. In their study, LV GLS remained essentially unchanged early after surgery but improved significantly by six months, simultaneously with a reduction in LV mass, whereas LVEF remained relatively unchanged [28]. Similarly, Chen et al. demonstrated progressive improvement in GLS after AVR in patients with AS, while patients with AR showed significant early deterioration at one week followed by recovery and marked improvement by three months [16]. These findings suggest that postoperative changes in strain depend on the underlying valvular lesion and the subsequent process of ventricular remodelling.

Perioperative GLS may also provide prognostic information beyond LVEF. Sonny et al. demonstrated that worse intraoperative GLS in patients undergoing AVR was associated with prolonged postoperative hospitalization (>7 days; 26% of patients), with each 1% worsening in GLS associated with higher odds of prolonged hospitalization (adjusted OR 1.22, 98.3% CI 1.01–1.47; $p = 0.012$), whereas LVEF was not significantly associated with the studied outcomes [29]. Wakefield et al. further showed that post-CPB GLS provided greater prognostic information than pre-CPB GLS or the relative change in GLS, with each 1% worsening in post-CPB deformation associated with prolonged hospitalization >7 days (adjusted OR 1.27, 97.5% CI 1.01–1.59; $p = 0.035$) and an increase in postoperative NT-proBNP of 98.4 pg/mL (98.3% CI 19.9–176.9; $p = 0.008$) [30]. These observations emphasize the importance of measurement timing, as post-CPB GLS may integrate the effects of the surgical intervention, CPB, and acute changes in loading conditions. However, prolonged hospitalization and postoperative NT-proBNP should be regarded as intermediate rather than hard clinical endpoints.

The CardioLink-2 subanalysis highlighted the importance of loading conditions when interpreting GLS after mitral valve surgery [17]. The cohort included 104 patients undergoing surgical repair for posterior leaflet prolapse. GLS deteriorated from −19.6% preoperatively to −12.8% immediately after repair and subsequently improved to approximately −16.0% at one year. Importantly, Rumman et al. showed that GLS indexed to LV end-diastolic volume returned to baseline at one year, suggesting that part of further impairment of GLS after mitral repair may reflect altered loading conditions rather than persistent intrinsic myocardial dysfunction [17]. This should also be interpreted according to MR etiology, since CardioLink-2 included primary degenerative MR, whereas secondary MR is associated with underlying LV myocardial disease and remodelling. In line with this concept, Kamperidis et al. demonstrated that after mitral valve repair of secondary MR the LV has immediate and short-term reverse remodelling and increase in forward stroke volume, although without improvement of LVEF or indexed GLS [31]. This early improvement of hemodynamics at discharge after a surgical repair is of paramount importance since it is associated with a favourable outcome [32].

Beyond the immediate postoperative period, persistent impairment of GLS may also provide prognostic information. Lee et al. studied 344 patients with surgically treated severe mitral valve disease, including 214 patients with primary MR and 130 with MS, who had preserved LVEF at follow-up 6 months to 2 years after surgery [33]. During follow-

up, 32 patients (9.3%) were hospitalized for worsening heart failure and 3 (0.8%) died from cardiac causes. They found that patients who subsequently experienced heart-failure hospitalization or cardiac death had significantly lower absolute postoperative LV GLS than those without events (12.1 ± 3.1% vs. 15.0 ± 3.2%, $p < 0.001$), whereas LVEF was comparable between groups (62.6 ± 5.9% vs. 63.3 ± 6.6%, $p = 0.550$). Moreover, postoperative |LV-GLS| ≤ 14% remained independently associated with these clinical events (adjusted HR 3.68, 95% CI 1.54–8.80; $p = 0.003$). Overall, changes in LV longitudinal mechanics after valve surgery appear to depend on the underlying valvular lesion, loading conditions, and timing of assessment. Early postoperative worsening of LV GLS, particularly after correction of regurgitant lesions, should therefore not be attributed solely to perioperative myocardial injury, as acute changes in ventricular loading or unloading may substantially influence myocardial deformation. Conversely, persistent GLS impairment during follow-up may identify residual myocardial dysfunction and provide prognostic information beyond LVEF.

6. Valve Surgery and Right Ventricular Longitudinal Strain

Assessment of RV function during valve surgery is particularly challenging because of the complex RV geometry and the substantial perioperative changes in loading conditions and contraction pattern. Conventional longitudinal indices such as TAPSE and RV S′ frequently deteriorate after open cardiac surgery, although global RV systolic performance may not change proportionally [34]. Speckle-tracking-derived RV longitudinal strain may therefore provide additional information on acute alterations in RV mechanics that are not fully captured by conventional parameters.

Duncan et al. provided an intraoperative assessment of RV deformation in patients undergoing surgical AVR for severe aortic stenosis [18]. In their study, myocardial deformation was assessed by transesophageal echocardiography before surgical incision and at the time after chest closure. In contrast to the improvement, they observed in LV systolic strain rate after AVR, RV longitudinal strain deteriorated by approximately 4.6% by the end of AVR surgery, accompanied by reductions in TAPSE and RV FAC. The mechanisms underlying this acute deterioration remain uncertain and may involve myocardial protection, altered septal mechanics, pericardial opening, changes in RV preload, and ventricular interdependence. Importantly, unlike the LV, the RV does not experience a comparable acute reduction in afterload after correction of aortic stenosis, which may partly contribute to the different ventricular response.

Supporting the concept that postoperative changes in longitudinal indices do not necessarily indicate global RV failure, Keyl et al. evaluated 20 patients undergoing surgical AVR and 20 undergoing transcatheter aortic valve implantation [35]. After surgical AVR, longitudinal RV contraction markedly decreased, whereas transverse contraction increased, while 3D-RVEF remained unchanged (55 ± 7% vs. 55 ± 6%, $p = 0.52$). This finding suggests that non-longitudinal components of RV contraction may contribute to preservation of global systolic performance in some surgical settings; however, this pattern has not been consistently demonstrated across studies and should not be inferred from preserved RVEF alone [35]. In contrast, no significant alteration in the RV contraction pattern was observed after transcatheter valve replacement, suggesting that the postoperative reduction in longitudinal shortening following open cardiac surgery may reflect a shift from longitudinal toward transverse RV contraction rather than a true deterioration in global RV systolic function. A similar alteration in RV longitudinal mechanics was demonstrated by Keller et al. in patients undergoing open-chest mitral valve surgery [19]. Using three-dimensional transesophageal echocardiography before and immediately after surgery, the investigators found that RV free-wall longitudinal strain deteriorated from −20.1 ± 7.1% to −15.4 ± 5.1% ($p < 0.001$), while TAPSE decreased from 13.1 ± 5.1 to 11.0 ± 3.5 mm ($p = 0.04$). Addition-

ally, the longitudinal contribution to RVEF also declined markedly, from 20 ± 5% to 13 ± 6% ($p < 0.001$). In contrast, the reduction in global RVEF was considerably less pronounced and did not reach statistical significance (37 ± 13% vs. 32 ± 9%, $p = 0.15$). These findings further support the concept that open mitral valve surgery predominantly alters the longitudinal component of RV contraction rather than producing a proportional decline in global RV systolic performance.

Importantly, these changes may persist beyond the immediate intraoperative period. A study by Orde et al. evaluated 158 patients undergoing mitral valve repair and demonstrated a marked deterioration in RV FWS from −22.6 ± 7% preoperatively to −15 ± 6% before hospital discharge ($p < 0.001$) [36]. The reduction was less pronounced after minimally invasive robotic repair compared with conventional open repair, and RV longitudinal function showed greater recovery at one year in the minimally invasive group, although it did not fully return to preoperative levels. Longer-term structural and functional RV remodelling after mitral surgery has also been demonstrated using three-dimensional echocardiography. Grapsa et al. reported significant reductions in RV end-diastolic volume and an improvement in RVEF six months after surgery for degenerative MR, with more favourable reverse RV remodelling after valve repair than replacement [37].

Overall, available evidence suggests that loss of RV longitudinal shortening is a common feature of open valve surgery but does not necessarily indicate a proportional deterioration in global RV systolic performance. The postoperative reduction in longitudinal deformation may reflect a redistribution of RV contraction as well as the combined effects of CPB, myocardial protection, changes in pericardial support and ventricular interaction rather than an isolated immediate effect of pericardiotomy, altered septal mechanics, and changes in loading conditions [38]. Consequently, perioperative RV strain should be interpreted in relation to the underlying valve lesion, surgical intervention, and prevailing hemodynamic conditions rather than as an isolated marker of myocardial injury.

Selected studies evaluating perioperative and longitudinal changes in LV and RV longitudinal strain after valve surgery are summarized in Table 2.

Table 2. Studies evaluating perioperative and longitudinal changes in left and right ventricular global longitudinal strain (GLS) after valve surgery.

Study/Year	Population/ Procedure (n)	Study Design/ Methodological Considerations	Strain Assessment & Software/Vendor	Main Findings
A. Left ventricular strain				
Rost et al., 2010 [28]	Severe AS undergoing AVR ($n = 33$); concomitant CABG $n = 14$	Longitudinal observational study; small final cohort; incomplete follow-up; mixed AVR ± CABG population	LV GLS; serial TTE; EchoPAC BT07, GE Healthcare (Vivid 7)	GLS remained unchanged early after AVR but improved significantly at 6 months, alongside LV mass reduction
Chen et al., 2015 [16]	Isolated AVR for severe AS ($n = 20$) or AR ($n = 16$); total $n = 36$	Longitudinal single-centre observational study; small cohort; short follow-up; image-quality dependence	LV GLS and GCS; TTE; 4D LV-Analysis 3.1, TomTec (Philips iE33 acquisition)	GLS improved progressively after AVR in AS; in AR, early deterioration was followed by marked recovery at 3 months
Sonny et al., 2018 [29]	AVR for AS ± CABG (GLS cohort $n = 86$)	Post hoc analysis of randomized-trial data; image-quality selection; limited sample; surrogate outcome	2D LV GLS; intraoperative TEE after induction; EchoPAC v112, GE Healthcare (Vivid S6/E9)	Worse GLS was associated with prolonged postoperative hospitalization > 7 days (OR 1.22 per 1% worsening, 98.3% CI 1.01–1.47), whereas LVEF was not

Table 2. *Cont.*

Study/Year	Population/ Procedure (n)	Study Design/ Methodological Considerations	Strain Assessment & Software/Vendor	Main Findings
Lee et al., 2021 [33]	Surgically treated severe mitral valve disease ($n = 344$)	Retrospective single-centre cohort; highly selected population; mixed MV pathology/procedures; few clinical events	2D postoperative LV GLS; TTE; Image Arena 4.6, TomTec, vendor-independent (GE Vivid E9/Philips iE33 acquisition)	Postoperative |GLS| ≤ 14% was independently associated with HF hospitalization or cardiac death (adjusted HR 3.68, 95% CI 1.54–8.80), despite preserved LVEF
Wakefield et al., 2022 [30]	Severe AS undergoing AVR ± CABG ($n = 72$)	Post hoc secondary analysis of randomized-trial data; image-quality selection; limited confounder adjustment; surrogate outcomes	LV GLS; intraoperative TEE pre- and post-CPB; EchoPAC v112, GE Healthcare	Worse post-CPB GLS was associated with prolonged hospitalization (OR 1.27 per 1% worsening, 97.5% CI 1.01–1.59) and higher postoperative NT-proBNP (+98.4 pg/mL per 1% worsening, 98.3% CI 19.9–176.9)
Rumman et al., 2026 [17]	Degenerative MR undergoing surgical repair ($n = 104$)	Post hoc multicentre randomized-trial subanalysis; selected well-compensated cohort; inter-site heterogeneity; 1-year follow-up	LV GLS; immediate post-repair to 1 year; blinded independent core-lab analysis; software/vendor not reported in the primary article	GLS deteriorated immediately after repair and partially recovered; GLS indexed to LVEDV returned to baseline at 1 year
B. Right ventricular mechanics/strain				
Keyl et al., 2016 [35]	Severe AS: surgical AVR ($n = 20$) vs. transfemoral TAVI ($n = 20$)	Prospective non-randomized pilot study; small groups; Heart-Team treatment allocation; potential residual confounding	RV longitudinal and transverse contraction; 3D RVEF; TTE; 4D RV-Function, TomTec (Philips iE33)	Surgical AVR reduced longitudinal contraction while transverse contraction increased; RVEF remained unchanged
Duncan et al., 2017 [18]	Severe AS undergoing surgical AVR ($n = 97$); RV strain $n = 54$)	Randomized-trial secondary analysis; substantial image-quality exclusions; heterogeneous surgical/myocardial-protection strategies	Intraoperative RV longitudinal strain; TEE; EchoPAC v112, GE Healthcare (Vivid S6/E9)	RV longitudinal strain deteriorated after AVR, with parallel reductions in TAPSE and RV FAC
Keller et al., 2020 [19]	Open-chest mitral valve surgery subgroup ($n = 20$)	Retrospective observational cohort; very small surgical subgroup; heterogeneous comparator populations; anesthetic/hemodynamic confounding	3D RV free-wall longitudinal strain; intraoperative TEE; 4D RV-Function 2.0, TomTec, with ReVISION for RVEFlong; Philips X7-2t acquisition	RV free-wall strain and longitudinal contribution to RVEF declined markedly, while global RVEF changed less
Orde et al., 2020 [36]	Mitral valve repair ($n = 158$); robotic minimally invasive ($n = 115$) vs. open sternotomy ($n = 43$)	Retrospective single-centre analysis; image-quality exclusions; non-randomized groups with baseline heterogeneity	RV free-wall strain; pre-discharge and 1 year; Syngo Velocity Vector Imaging, Siemens, vendor-agnostic	RV strain deteriorated after surgery; impairment was less pronounced and recovery greater after minimally invasive repair

Abbreviations: AR: aortic regurgitation; AS: aortic stenosis; AVR: aortic valve replacement; CABG: coronary artery bypass grafting; CI: confidence interval; CPB: cardiopulmonary bypass; FAC: fractional area change; GCS: global circumferential strain; GLS: global longitudinal strain; HF: heart failure; HR: hazard ratio; LV: left ventricle; LVEDV: left ventricular end-diastolic volume; LVEF: left ventricular ejection fraction; MR: mitral regurgitation; MV: mitral valve; NT-proBNP: N-terminal pro-B-type natriuretic peptide; OR: odds ratio; RV: right ventricle; RVEF: right ventricular ejection fraction; RVFWLS: right ventricular free-wall longitudinal strain; TAPSE: tricuspid annular plane systolic excursion; TAVI: transcatheter aortic valve implantation; TEE: transesophageal echocardiography; TTE: transthoracic echocardiography; 2D: two-dimensional; 3D: three-dimensional.

7. Effect of General Anesthesia and Mechanical Ventilation on Myocardial Deformation

General anesthesia and IPPV substantially alter the physiological conditions under which perioperative echocardiographic measurements are obtained. As emphasized by Tol et al., commonly used intravenous and inhalational anesthetic agents exert dose-dependent effects on myocardial contractility, vascular tone, heart rate, and cardiac output, thereby modifying preload and afterload [20]. These effects are further influenced by IPPV, which increases intrathoracic pressure, reduces systemic venous return and RV preload, may increase PVR and RV afterload, and reduces LV preload, while simultaneously decreasing LV afterload. Consequently, the transition from the awake, spontaneously breathing state to general anesthesia with mechanical ventilation represents a major change in the loading environment of both ventricles.

This is particularly relevant for deformation imaging because myocardial strain is influenced by loading conditions. Experimental and clinical alterations in preload and afterload can modify longitudinal strain even in the absence of intrinsic changes in myocardial contractility [8]. Accordingly, awake preoperative and post-induction intraoperative strain measurements should not be considered physiologically equivalent, and differences between these time points cannot automatically be attributed to the surgical intervention or CPB.

7.1. Effect on Left Ventricular Global Longitudinal Strain

Available evidence indicates that LV longitudinal deformation may deteriorate after induction of general anesthesia even when conventional measures of systolic function remain preserved. Yang et al. demonstrated that propofol induction in patients with normal baseline LV function reduced longitudinal strain from approximately −22.3% to −19.4%, together with reductions in mitral annular systolic and diastolic velocities, whereas LVEF remained unchanged [39]. This finding is consistent with the observations highlighted by Tol et al., showing that general anesthesia may reduce stroke volume, cardiac output, and LV GLS while LVEF frequently remains preserved [20].

However, changes in myocardial deformation after propofol administration likely reflect more than a direct negative inotropic effect. Coleman et al. in their study evaluated spontaneously breathing patients receiving propofol sedation and found no significant deterioration in GLS despite a substantial reduction in arterial pressure, while LVEF also remained unchanged [40]. These findings suggest that changes in myocardial deformation reflect the combined effects of anesthetic exposure, ventilation strategy, and altered ventricular loading. Accordingly, studies performed during spontaneous ventilation provide more direct information on anesthetic exposure itself [40], whereas studies combining general anesthesia with positive-pressure ventilation assess the integrated effects of anesthesia and altered loading conditions [21].

Extending these observations, Dalla et al. evaluated the effects of general anesthesia and mechanical ventilation on myocardial deformation in patients without known myocardial disease [21]. The authors noted that LV GLS deteriorated from −19.1 ± 2.3% while patients were awake and spontaneously breathing to −17.3 ± 2.9% after induction, intubation, and initiation of mechanical ventilation. This occurred in parallel with reductions in mean arterial pressure, stroke volume index, cardiac index, and LV end-diastolic volume, while LV end-systolic elastance decreased by approximately 23%. These findings suggest that the observed deterioration in deformation reflected a combination of altered loading conditions and reduced contractile state rather than a single isolated mechanism. The dynamic and potentially reversible nature of these changes was further highlighted by Cinotti et al. [41]. More specifically, median LV GLS deteriorated from approximately

−20.6% in awake patients to −18.2% after induction and mechanical ventilation despite preserved LVEF, but returned toward baseline after extubation.

The choice of anesthetic technique may also modify perioperative longitudinal mechanics, although available evidence remains inconsistent. In patients undergoing on-pump CABG, Chennakeshavallu et al. observed deterioration in LV GLS with both sevoflurane- and propofol-based anesthesia [42]. The study showed that there was no significant between-group difference in global LV GLS after CPB, although some regional longitudinal strain parameters appeared better preserved with sevoflurane. In a randomized study of patients undergoing off-pump CABG, Gong et al. reported more preserved LV GLS with the use of sevoflurane than with propofol-based total intravenous anesthesia at selected intraoperative time points [43]. However, anesthetic effects in these studies cannot be fully separated from changes related to revascularization, surgical manipulation, loading conditions, and, in the on-pump setting, ischemia–reperfusion and CPB. Therefore, current evidence does not establish a consistent superiority of one anesthetic technique for preservation of myocardial deformation [20,42,43]. In the cardiac-surgical studies, differences in ventilatory settings, PEEP, vasoactive support, and surgical or CPB-related hemodynamic changes further limit attribution of strain changes to the anesthetic agent alone.

Anesthetic depth has been proposed as a potential modifier of perioperative myocardial deformation, although direct evidence supporting such an effect is currently lacking. The dose-dependent cardiovascular effects of anesthetic agents provide a physiological rationale for differences in anesthetic exposure to influence the hemodynamic conditions under which strain is measured. Puri and Murthy showed that BIS-guided titration during cardiac surgery reduced episodes of hypertension and tachycardia and facilitated adjustment of anesthetic delivery [44]. Similarly, Chiu et al. randomized 20 patients undergoing hypothermic CPB to conventional propofol administration or titration to a BIS target of 40–50 and demonstrated substantially lower propofol requirements with BIS-guided administration [45]. However, these studies were not designed to evaluate myocardial deformation, while strain studies incorporating BIS monitoring have generally not compared predefined levels of anesthetic depth. Therefore, whether anesthetic depth independently influences LV or RV deformation remains uncertain. Prospective studies using predefined anesthetic-depth targets, standardized ventilatory and hemodynamic conditions, and serial perioperative strain assessment are needed to distinguish the effects of anesthetic exposure from those of loading changes, CPB, and the surgical intervention itself.

Overall, the available evidence indicates that LV GLS may deteriorate after induction of general anesthesia and mechanical ventilation even in the absence of surgical intervention, while LVEF frequently remains relatively unchanged. The magnitude and direction of this response are not uniform and reflect the combined influences of myocardial contractility, vascular tone, preload, afterload, anesthetic exposure, and ventilatory conditions. Consequently, differences between awake preoperative and post-induction intraoperative LV GLS should not automatically be interpreted as newly developed myocardial dysfunction.

7.2. Effect on Right Ventricular Longitudinal Strain

The right ventricle may be particularly sensitive to the transition from spontaneous breathing to IPPV because its performance is strongly dependent on venous return and pulmonary vascular loading. Reduced venous return may decrease RV preload, whereas increases in PVR and intrathoracic pressure may increase RV afterload. Nevertheless, as highlighted by Tol et al., the available literature on the effects of general anesthesia and IPPV on RV longitudinal deformation is less extensive and more heterogeneous than that for the LV [20].

Dalla et al. demonstrated deterioration in RV FWS from −26.5 ± 3.9% while patients were awake and spontaneously breathing to −24.1 ± 4.2% after induction of anesthesia and mechanical ventilation [21]. Also, RV end-diastolic area decreased, supporting an important contribution of altered ventricular filling to the observed change in longitudinal deformation.

A similar dissociation between longitudinal and global RV function was demonstrated by Magunia et al. in patients undergoing elective cardiac surgery [46]. Following induction of general anesthesia and initiation of positive-pressure ventilation, TAPSE, RV S′, and RV longitudinal strain decreased significantly, whereas RVEF, RV stroke volume, and indices of RV volume remained relatively preserved. This pattern is similar to that observed after CABG and valve surgery and emphasizes that deterioration in longitudinal RV indices does not necessarily represent a proportional decline in global RV systolic performance.

The influence of ventilatory loading on RV deformation is further illustrated by Franchi et al. [47]. In this study, increasing PEEP from 5 to 15 cmH_2O was associated with deterioration in RV longitudinal strain from approximately −20.2% to −16.3%, accompanied by reductions in stroke volume and cardiac output, whereas LV longitudinal strain remained relatively unchanged. Although this study involved mechanically ventilated critically ill patients rather than elective cardiac surgical patients, it provides mechanistic evidence that RV longitudinal deformation is particularly sensitive to changes in intrathoracic pressure and pulmonary vascular loading. However, the magnitude and clinical significance of changes in RV strain related to general anesthesia and IPPV remain incompletely established [20].

Selected studies evaluating the effects of general anesthesia and mechanical ventilation on LV and RV longitudinal strain are summarized in Table 3.

Table 3. Studies evaluating perioperative changes in left and right ventricular longitudinal strain during general anesthesia and mechanical ventilation.

Study/Year	Population/ Setting (n)	Study Design/Key Limitations	Anesthetic/Ventilatory Exposure and Strain Assessment/ Software/Vendor	Main Findings
A. Left ventricular strain				
Yang et al., 2013 [39]	Normal LV function undergoing non-cardiac surgery ($n = 19$)	Prospective single-centre observational study; small, all-female low-risk cohort	Propofol induction; LV longitudinal strain; TTE; EchoPAC v10, GE Healthcare (Vivid Q)	Longitudinal strain deteriorated from ~−22.3% to −19.4%, while LVEF remained unchanged
Dalla et al., 2019 [21]	Patients without myocardial disease ($n = 21$)	Small observational study; highly selected healthy cohort; anesthesia and PPV effects not separable	Awake spontaneous breathing vs. GA + positive-pressure ventilation; 2D LV GLS; TTE; EchoPAC v201, GE Healthcare (Vivid E9)	LV GLS deteriorated from −19.1% to −17.3%, along with reductions in MAP, SVI, CI, and LVEDV
Cinotti et al., 2020 [41]	Non-cardiac surgical patients ($n = 27$)	Prospective single-centre pilot study; small selected cohort; image-quality exclusions	Awake, post-induction/MV, preload modification, and post-extubation; 2D LV GLS; TTE; EchoPAC, GE Healthcare (Vivid S6)	GLS deteriorated after GA/MV from −20.6% to −18.2%, remained impaired after passive leg raising (−17.6%), and returned to baseline after extubation (−21.7%); LVEF remained unchanged

Table 3. *Cont.*

Study/Year	Population/ Setting (n)	Study Design/Key Limitations	Anesthetic/Ventilatory Exposure and Strain Assessment/ Software/Vendor	Main Findings
Coleman et al., 2021 [40]	Patients undergoing elective outpatient endoscopic procedures ($n = 16$)	Prospective single-centre pilot study; very small sample; non-standardized propofol dosing; unblinded analysis	Propofol sedation with spontaneous ventilation; 2D LV GLS; TTE; AutoSTRAIN LV, Philips Medical Systems (EPIQ CVxi)	No significant deterioration in GLS (−14.6% to −15.8%, $p = 0.20$) despite reduced arterial pressure; LVEF remained unchanged
Chennake-shavallu et al., 2022 [42]	On-pump CABG with normal LV function ($n = 50$)	Prospective non-randomized study; small selected cohort; image-quality exclusions; CPB-related confounding	Sevoflurane vs. propofol; baseline TTE and pre-/post-CPB TEE; 2D LV GLS/segmental PSLS; QLAB 9, Philips (iE33)	GLS deteriorated with both techniques; no significant between-group difference in global GLS after CPB
Gong et al., 2024 [43]	Off-pump CABG; randomized $n = 60$, analyzed $n = 55$ (sevoflurane $n = 29$; TIVA $n = 26$)	Prospective randomized trial; modest sample; post-randomization exclusions; short-term assessment	Sevoflurane vs. propofol-based TIVA; serial intraoperative TEE; LV GLS; QLab 10.7/aCMQ, Philips Healthcare (EPIQ 7C)	GLS was better preserved with sevoflurane at selected intraoperative time points
B. Right ventricular strain				
Franchi et al., 2013 [47]	Mechanically ventilated critically ill patients ($n = 20$)	Prospective observational study; very small heterogeneous ICU cohort; sequential PEEP exposure	PEEP 5–15 cmH_2O; RV longitudinal strain; TTE; XStrain/MyLab 70 Xvision, Esaote	RV strain deteriorated from ~−20.2% to −16.3% at higher PEEP, with reductions in stroke volume and cardiac output
Dalla et al., 2019 [21]	Patients without myocardial disease ($n = 21$)	Small observational study; highly selected healthy cohort; anesthesia and PPV effects not separable	Awake spontaneous breathing vs. GA + positive-pressure ventilation; RV free-wall strain; TTE; EchoPAC v201, GE Healthcare (Vivid E9)	RV free-wall strain deteriorated from −26.5% to −24.1%, accompanied by reduced RV filling
Magunia et al., 2019 [46]	Patients undergoing elective cardiac surgery ($n = 53$)	Prospective single-centre observational study; heterogeneous cardiac-surgery cohort; modest sample	Before vs. after GA induction and positive-pressure ventilation; RV free-wall longitudinal strain; TTE; 4D RV-Function 2.0/Image Arena, TomTec (Philips iE33)	TAPSE, RV S′, and RV free-wall longitudinal strain decreased from −22.9 ± 6.0% to −21.4 ± 5.9% ($p = 0.0366$), while RVEF and RV stroke volume remained relatively preserved

Abbreviations: CABG: coronary artery bypass grafting; CI: cardiac index; CPB: cardiopulmonary bypass; GA: general anesthesia; GLS: global longitudinal strain; ICU: intensive care unit; LV: left ventricle; LVEDV: left ventricular end-diastolic volume; LVEF: left ventricular ejection fraction; MAP: mean arterial pressure; MV: mechanical ventilation; PEEP: positive end-expiratory pressure; PPV: positive-pressure ventilation; PSLS: peak systolic longitudinal strain; RV: right ventricle; RVEF: right ventricular ejection fraction; RV S′: systolic tricuspid annular velocity; SVI: stroke volume index; TAPSE: tricuspid annular plane systolic excursion; TEE: transesophageal echocardiography; TIVA: total intravenous anesthesia; TTE: transthoracic echocardiography; 2D: two-dimensional.

8. Limitations of the Evidence and Methodological Considerations

The available evidence on perioperative ventricular strain in cardiac surgery should be interpreted in the context of several important limitations. Most studies included in this

narrative review are relatively small, single-centre observational cohorts or pilot studies, and several comprise fewer than 50 patients. Consequently, many reported associations and perioperative strain trajectories should be regarded as hypothesis-generating rather than definitive evidence of causal effects of surgery, cardiopulmonary bypass, anesthesia, or mechanical ventilation. Furthermore, some studies included heterogeneous cardiac-surgical populations, whereas others focused exclusively on CABG or specific valve procedures, limiting direct comparison across cohorts.

Moreover, substantial methodological heterogeneity also exists between studies. Myocardial deformation has been assessed using different imaging modalities and acquisition conditions, including transthoracic and transesophageal echocardiography, two- and three-dimensional speckle-tracking techniques, and different software packages and vendors [48,49].

Image quality and tracking reliability represent additional sources of measurement uncertainty, particularly for RV strain and intraoperative transesophageal echocardiography, and several studies excluded patients or myocardial segments because of inadequate image quality [15,22]. Where formally assessed, reproducibility was generally good; for example, Howard-Quijano et al. reported intraobserver and interobserver intraclass correlation coefficients of 0.92 and 0.91, respectively, for postoperative 3D longitudinal strain [7].

Overall, differences in equipment, software, imaging modality, strain definition, and acquisition conditions limit comparison of absolute strain values between studies. Given the recognized inter-vendor variability of strain measurements, serial GLS assessments should, where possible, be performed using the same equipment and analysis software [48,49].

9. Implications for Practice and Future Research

In clinical practice, perioperative left and right ventricular strain should be interpreted as a complementary parameter of ventricular functional response to anesthesia, mechanical ventilation, CPB and cardiac surgery. An isolated deterioration in post-CPB GLS should not be considered as definite evidence of myocardial injury, but should be interpreted alongside the other parameters of ventricular function, regional wall motion abnormalities, loading conditions, vasoactive and inotropic support, type of mechanical ventilation, and the underlying treated disease [2,20]. GLS is a load-dependent variable and has to be adjusted to the afterload that the ventricle faces at each time point. At the perioperative period, the ventricular afterload changes at every stage of the procedure. Hence, data about the echocardiography evaluated myocardial work based on pressure-strain loops are in need for the perioperative period to further assess in depth the ventricular mechanics. Left and right ventricular strain changes dynamically post-intervention, to a varied extent, that has to be further studied. It would be of paramount clinical importance for the future studies to identify the parameters leading to ventricular mechanics recovery post-surgical intervention. Although available studies suggest that perioperative GLS provides prognostic information beyond LVEF and, in some cohorts, even beyond conventional clinical risk factors [29,30,34], strain-guided perioperative management has not yet been shown to improve clinical outcomes [22]. However, the current evidence primarily supports strain for perioperative risk stratification rather than therapeutic guidance, while its incremental value beyond established clinical risk scores, biomarkers, and conventional echocardiography remains to be established. Moreover, evidence remains substantially stronger for LV GLS than for RV strain, for which standardization and prospective validation are still limited [22]. Further clinical studies should be conducted to determine whether clinically actionable perioperative strain thresholds can be established to guide the perioperative medical management and the patient's follow-up plan and future prognosis.

10. Conclusions

In conclusion, perioperative strain should be interpreted as a dynamic measure of myocardial mechanics obtained under specific physiological conditions rather than as a load-independent marker of myocardial contractility. General anesthesia and mechanical ventilation can alter both LV and RV longitudinal deformation before the surgical intervention itself through changes in contractility, vascular tone, ventricular loading, and intrathoracic pressure. Anesthetic technique may contribute to variability in perioperative strain, whereas the potential influence of anesthetic depth remains a hypothesis requiring further investigation. Therefore, comparisons between preoperative, post-induction, post-CPB, and postoperative strain measurements should be interpreted in relation to the prevailing hemodynamic profile, ventilator settings, anesthetic exposure, vasoactive support, and timing of acquisition before changes are attributed to CPB or perioperative myocardial injury.

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Informed Consent Statement: Written informed consent was obtained from the patient whose echocardiographic images are presented in Figure 1 for the use of their fully de-identified clinical data and imaging material for research and scientific publication purposes. No patient-identifying information is displayed.

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Abbreviations

The following abbreviations are used in this manuscript:

Abbreviation	Definition
AR	Aortic regurgitation
AS	Aortic stenosis
AVR	Aortic valve replacement
BIS	Bispectral index
CABG	Coronary artery bypass grafting
CI	Confidence interval

CPB	Cardiopulmonary bypass
FAC	Fractional area change
FWS	Free-wall strain
HR	Hazard ratio
GLS	Global longitudinal strain
ICU	Intensive care unit
IPPV	Intermittent positive-pressure ventilation
IQR	Interquartile range
LV	Left ventricle/left ventricular
LV S′	Mitral annular systolic velocity
LVEF	Left ventricular ejection fraction
MAG	Multiple arterial grafting
MR	Mitral regurgitation
MS	Mitral stenosis
NT-proBNP	N-terminal pro-B-type natriuretic peptide
OR	Odds ratio
PEEP	Positive end-expiratory pressure
PVR	Pulmonary vascular resistance
RV	Right ventricle/right ventricular
RVEF	Right ventricular ejection fraction
RV S′	Systolic tricuspid annular velocity
SAG	Single arterial grafting
STE	Speckle-tracking echocardiography
TAPSE	Tricuspid annular plane systolic excursion

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