Preprints.org

Review

Not peer-reviewed version

Epicardial and Visceral Adipose Tissue and Global Longitudinal Strain: A Review of Cardiac Imaging Insights in Subclinical Myocardial Dysfunction

Marco Vicardi , Afshin Farzaneh-Far , Cristiano Fava , Luca Dalle Carbonare , Simone Romano *

Posted Date: 12 February 2026

doi: 10.20944/preprints202602.0976.v1

Keywords: epicardial adipose tissue; visceral adipose tissue; global longitudinal strain; speckle-tracking echocardiography; subclinical cardiovascular disease; mediterranean diet

Preprints.org is a free multidisciplinary platform providing preprint service that is dedicated to making early versions of research outputs permanently available and citable. Preprints posted at Preprints.org appear in Web of Science, Crossref, Google Scholar, Scilit, Europe PMC.

Copyright: This open access article is published under a Creative Commons CC BY 4.0 license, which permit the free download, distribution, and reuse, provided that the author and preprint are cited in any reuse.

Preprints.org (www.preprints.org) | NOT PEER-REVIEWED | Posted: 12 February 2026

doi:10.20944/preprints202602.0976.v1

Disclaimer/Publisher's Note: The statements, opinions, and data contained in all publications are solely those of the individual author(s) and contributor(s) and not of MDPI and/or the editor(s). MDPI and/or the editor(s) disclaim responsibility for any injury to people or property resulting from any ideas, methods, instructions, or products referred to in the content.

Review

Epicardial and Visceral Adipose Tissue and Global Longitudinal Strain: A Review of Cardiac Imaging Insights in Subclinical Myocardial Dysfunction

Marco Vicardi [1], Afshin Farzaneh-Far [2], Cristiano Fava [1], Luca Dalle Carbonare [1] and Simone Romano [1]

[1] Unit of General Medicine C, Medicine Department, University of Verona and University and Hospital Trust (AOUI) of Verona, Verona, Italy

[2] Division of Cardiology, Department of Medicine, Duke University, Durham, North Carolina

* Correspondence: simone.romano@univr.it

Abstract

Background: Visceral adipose tissue (VAT) and epicardial adipose tissue (EAT) are increasingly recognized as relevant contributors to cardiometabolic alterations and subclinical myocardial dysfunction, independently of overall obesity. Their pathogenic role extends beyond simple fat accumulation, encompassing inflammatory activation, lipotoxicity, and altered myocardial metabolism. **Objective:** This narrative review synthesizes current evidence on the relationships between VAT/EAT and myocardial strain parameters, with emphasis on subclinical cardiovascular risk detection and nutritional interventions. **Methods:** We conducted a comprehensive review of studies published between 2003-2025, focusing on imaging-based assessments of adipose tissue distribution and strain parameters using echocardiography, computed tomography, and cardiac magnetic resonance. **Results:** Increased EAT, and to a lesser extent VAT, showed significant associations with impaired global longitudinal strain (GLS) across imaging-based studies. In patients with type 2 diabetes, VAT mediated a substantial proportion of the association between insulin resistance and left ventricular dysfunction. Mediterranean diet adherence was associated with reduced EAT burden and lower prevalence of persistent atrial fibrillation in selected populations. Speckle-tracking echocardiography consistently showed superior prognostic value compared to ejection fraction for detecting subclinical dysfunction. **Conclusions:** VAT and EAT represent important mechanistic links between body composition and early myocardial dysfunction, identifiable through advanced strain imaging before clinical disease becomes apparent. These findings support the integration of multimodal cardiac imaging and nutritional interventions into cardiovascular prevention strategies, providing novel opportunities for early risk stratification and personalized treatment approaches.

Keywords: epicardial adipose tissue; visceral adipose tissue; global longitudinal strain; speckle-tracking echocardiography; subclinical cardiovascular disease; mediterranean diet

---

1. Introduction

The Paradigm Shift in Cardiovascular Risk Assessment

Contemporary cardiovascular medicine is progressively moving beyond traditional risk factor–based approaches toward more refined phenotyping strategies that integrate imaging biomarkers and body composition analysis [1]. This evolution reflects growing recognition that standard risk calculators, while valuable, may incompletely reflect the complexity of individual cardiovascular risk profiles, particularly in metabolically heterogeneous populations.

© 2025 by the author(s). Distributed under a Creative Commons CC BY license.

Preprints.org (www.preprints.org) | NOT PEER-REVIEWED | Posted: 12 February 2026 doi:10.20944/preprints202602.0976.v1

The limitations of body mass index (BMI) as a cardiovascular risk predictor have become increasingly apparent, with substantial evidence demonstrating that adipose tissue distribution patterns provide superior prognostic information compared to total body weight measures [2]. This recognition has catalyzed intensive research into "ectopic fat" depots—adipose tissue accumulations in non-traditional locations that exert profound metabolic and cardiovascular effects through proximity-dependent and systemic mechanisms.

Visceral adipose tissue (VAT) and epicardial adipose tissue (EAT) have emerged as particularly relevant components of cardiovascular risk assessment and as potential targets of preventive strategies [1,3]. Unlike subcutaneous fat, these visceral depots exhibit heightened metabolic activity and inflammatory potential, directly influencing cardiovascular pathophysiology through paracrine, vasocrine, and systemic pathways [1]. The anatomical proximity of these fat depots to cardiac structures creates unique opportunities for both pathogenic interactions and therapeutic targeting.

Rationale for Focus on VAT and EAT

Visceral adipose tissue, defined as intra-abdominal fat surrounding internal organs, has been extensively studied for its role in metabolic syndrome and cardiovascular disease [4]. VAT demonstrates distinct characteristics compared to subcutaneous fat, including enhanced lipolytic activity, increased inflammatory cytokine production, and direct portal circulation connections that bypass first-pass hepatic metabolism. These properties position VAT as a critical mediator of insulin resistance, dyslipidemia, and systemic inflammation [4,5].

Epicardial adipose tissue represents an even more anatomically intimate cardiovascular risk factor [1,2]. Located between the myocardium and visceral pericardium, EAT shares microcirculation with the underlying myocardium and lacks fascial boundaries that would limit paracrine interactions [1]. This unique anatomical arrangement enables direct tissue-to-tissue communication through local release of adipokines, inflammatory mediators, and vasoactive substances [1].

The physiological functions of EAT under normal conditions include thermogenesis, mechanical cardiac protection, and metabolic buffering [1,6]. However, pathological EAT expansion and inflammatory activation transform this protective tissue into a source of cardiotoxic mediators that are associated with coronary atherosclerosis, myocardial fibrosis, and electrical remodeling [7,8].

Strain Imaging as Early Marker of Dysfunction

Speckle-tracking echocardiography (STE) has revolutionized the detection of subclinical cardiovascular dysfunction by enabling quantitative assessment of myocardial deformation parameters that precede changes in ejection fraction [9,10]. Global longitudinal strain (GLS) has emerged as the most robust and clinically validated strain parameter, consistently demonstrating superior sensitivity for detecting early systolic dysfunction and providing incremental prognostic value across diverse patient populations. [9,10]

GLS has been consistently associated with prognostic outcomes across a wide spectrum of cardiovascular conditions and selected non-cardiovascular diseases, as demonstrated using multimodal cardiac imaging approaches, including echocardiography and cardiac magnetic resonance [11–24]

The technical advancement from tissue Doppler-based strain measurements to speckle-tracking methodologies has substantially improved reproducibility and clinical applicability [10,25]. Contemporary strain analysis can detect subtle alterations in myocardial mechanics that occur months to years before conventional echocardiographic abnormalities become apparent, creating unprecedented opportunities for early intervention and prevention [10].

Strain imaging applications extend beyond left ventricular assessment to include the evaluation of atrial mechanics [26]. Left atrial strain, in particular, has gained recognition as a sensitive marker of elevated filling pressures and predictor of atrial fibrillation development, with direct relevance to patients with increased visceral adiposity [26].

© 2025 by the author(s). Distributed under a Creative Commons CC BY license.

Preprints.org (www.preprints.org) | NOT PEER-REVIEWED | Posted: 12 February 2026 doi:10.20944/preprints202602.0976.v1

Review Objectives and Scope

This comprehensive review synthesizes current evidence linking VAT and EAT to myocardial strain abnormalities, with particular emphasis on subclinical cardiovascular risk detection and therapeutic implications. Our analysis encompasses carefully selected studies spanning foundational research, clinical investigations, and intervention trials published between 2003 and 2025.

The review addresses several critical knowledge gaps in the field: first, the comparative importance of VAT versus EAT in predicting strain abnormalities; second, the mechanistic pathways linking adipose tissue dysfunction to myocardial performance; third, the clinical utility of combined imaging approaches for risk stratification; and fourth, the evidence supporting nutritional and lifestyle interventions for modifying both adipose tissue characteristics and strain parameters.

Special attention is devoted to nutritional interventions, reflecting growing interest in dietary pattern modifications as first-line therapeutics for cardiovascular prevention [23,24]. The Mediterranean diet, in particular, has demonstrated significant effects on both adipose tissue biology and cardiovascular outcomes, making it highly relevant for integrated prevention strategies that combine imaging biomarkers with lifestyle modifications [23,24] (Figure 1).

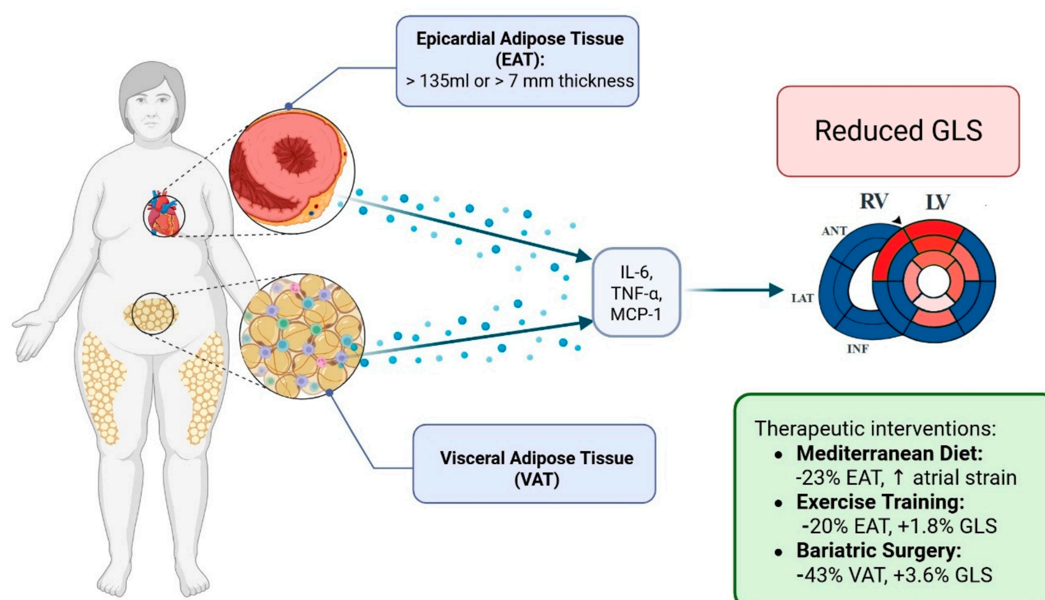

Figure 1. Conceptual overview of the association between epicardial and visceral adipose tissue and subclinical myocardial dysfunction. Increased epicardial adipose tissue (EAT) and visceral adipose tissue (VAT) are associated with a pro-inflammatory profile that correlates with impaired myocardial deformation, expressed as reduced left ventricular global longitudinal strain (GLS), highlighted in red in the figure. Lifestyle and weight-loss interventions are associated with reductions in EAT/VAT and parallel improvements in myocardial strain.

2. Adipose Tissue Biology and Cardiovascular Pathophysiology

Visceral Adipose Tissue: Beyond Energy Storage

Visceral adipose tissue represents far more than a passive energy storage depot, functioning as a highly active endocrine organ that profoundly influences cardiovascular health through multiple interconnected pathways [4,27]. The anatomical distribution of VAT encompasses intra-abdominal fat surrounding the liver, pancreas, and intestines, with direct venous drainage into the portal circulation that bypasses hepatic first-pass metabolism and enables immediate systemic effects of secreted mediators [4,5,27].

© 2025 by the author(s). Distributed under a Creative Commons CC BY license.

Preprints.org (www.preprints.org) | NOT PEER-REVIEWED | Posted: 12 February 2026 doi:10.20944/preprints202602.0976.v1

The inflammatory secretome of VAT includes a complex array of cytokines, chemokines, and adipokines that collectively promote cardiovascular dysfunction [27,28]. Key inflammatory mediators include tumor necrosis factor-alpha (TNF-α), interleukin-6 (IL-6), monocyte chemoattractant protein-1 (MCP-1), and C-reactive protein (CRP). In patients with type 2 diabetes, VAT demonstrates positive associations with insulin resistance and negative associations with left ventricular GLS [27].

Metabolic dysfunction in expanded VAT manifests through enhanced lipolysis, increased free fatty acid (FFA) release, and impaired insulin sensitivity [27]. This metabolic dysregulation creates a systemic environment characterized by lipotoxicity and inflammation that is associated with alterations in myocardial metabolism and function [27]. The relationship between VAT and insulin resistance is particularly relevant, as insulin resistance mediates approximately 60-65% of the association between visceral adiposity and left ventricular dysfunction [27].

The progression from metabolic stress to structural cardiovascular changes involves complex pathophysiological mechanisms. Enhanced FFA availability alters myocardial substrate utilization, shifting from glucose toward fatty acid oxidation and reducing cardiac efficiency [29,30]. Simultaneously, chronic inflammation promotes coronary endothelial dysfunction, microvascular rarefaction, and interstitial fibrosis that collectively impair myocardial mechanics [27,29,31].

Epicardial Adipose Tissue: The Heart's Local Fat Depot

Epicardial adipose tissue possesses unique anatomical and functional characteristics that distinguish it from other visceral fat depots and create particularly intimate cardiovascular interactions [1,31]. The absence of fascial barriers between EAT and the underlying myocardium enables direct paracrine signaling, while shared microcirculation facilitates bidirectional communication between adipocytes and cardiomyocytes [1,31].

Under physiological conditions, EAT serves several protective functions including thermogenesis through brown adipose tissue-like activity, mechanical cushioning during cardiac contraction, and metabolic buffering that provides local energy substrates during periods of high cardiac demand [1,6,32]. The expression of uncoupling proteins and presence of multilocular adipocytes in healthy EAT support its thermogenic capacity and potential cardioprotective role [6,32].

However, pathological EAT expansion transforms this beneficial tissue into a source of cardiotoxic mediators [7,28,32]. Inflammatory activation of EAT leads to increased production of pro-inflammatory cytokines including TNF-α, IL-6, and IL-1β, while simultaneously reducing protective adipokine secretion such as adiponectin [7,28]. This inflammatory shift creates a local environment that promotes coronary atherosclerosis, myocardial fibrosis, and electrical remodeling [7].

Regional heterogeneity within EAT adds additional complexity to its cardiovascular effects [1,33]. Pericoronary fat demonstrates distinct inflammatory characteristics compared to periatrial or periventricular EAT, with enhanced expression of inflammatory markers in regions adjacent to coronary atherosclerotic plaques [33]. This spatial relationship suggests that EAT may both contribute to and respond to local coronary pathology, creating self-reinforcing cycles of inflammation and dysfunction [1,33].

Mechanistic Links to Myocardial Dysfunction

The pathways connecting adipose tissue dysfunction to impaired myocardial strain involve both direct tissue effects and indirect systemic mechanisms that ultimately converge on altered cardiomyocyte function and extracellular matrix composition [31]. Direct effects include lipotoxicity from excessive FFA delivery, local inflammatory mediator release, and paracrine signaling that influences cardiomyocyte metabolism and contractile protein function [31].

Lipotoxicity represents a relevant mechanistic pathway whereby excessive fatty acid availability overwhelms myocardial oxidative capacity, leading to intracellular lipid accumulation, ceramide production, and mitochondrial dysfunction [29,30,34]. This metabolic stress impairs excitation-

© 2025 by the author(s). Distributed under a Creative Commons CC BY license.

contraction coupling, reduces contractile protein sensitivity, and promotes cardiomyocyte apoptosis, all of which contribute to reduced strain parameters before changes in ejection fraction become apparent [35].

Epicardial adipose tissue serves as a significant source of proinflammatory mediators including IL-1β, IL-6, MCP-1, and TNF-α [28]. In pathological conditions, EAT transitions from a protective to a deleterious tissue, secreting adipokines that promote myocardial fibrosis and structural remodeling [31]. This inflammatory-driven remodeling contributes to increased myocardial stiffness and functional impairment, effects that are particularly evident in longitudinal strain parameters due to the subendocardial vulnerability of longitudinally oriented muscle fibers [31,36]. The relationship between EAT and myocardial dysfunction is reflected in inverse correlations demonstrated between EAT volume and both global longitudinal and circumferential strain in heart failure patients [31]

Coronary microvascular dysfunction represents an often-overlooked pathway linking adipose tissue inflammation to myocardial dysfunction [8,29,37]. VAT and EAT-derived inflammatory mediators promote endothelial dysfunction, reduce nitric oxide bioavailability, and impair coronary flow reserve [37]. These microvascular alterations may contribute to regional variations in myocardial perfusion, potentially reflected by heterogeneous strain patterns even in the absence of epicardial coronary disease [8,29,37].

The temporal progression from metabolic stress to structural changes follows predictable patterns that can be monitored through serial strain assessments [36]. Early changes include reduced strain rate parameters reflecting impaired diastolic relaxation, followed by reductions in peak systolic strain values, and finally the development of regional strain heterogeneity that reflects evolving fibrosis patterns [36]. This predictable sequence creates opportunities for intervention through serial strain assessments before irreversible structural changes occur.

3. Speckle-Tracking Echocardiography: Technical Foundations

Physics and Technical Principles

Speckle-tracking echocardiography represents a significant advancement in cardiac imaging capability, enabling quantitative assessment of myocardial deformation [9,10,36]. The fundamental physics underlying STE involves analysis of frame-to-frame displacement of tissue speckles created by constructive and destructive interference patterns from ultrasound reflection at the tissue interface level [9,36,38].

Contemporary STE systems employ advanced pattern recognition algorithms that can track individual speckle patterns with high spatial resolution, depending on image quality and vendor-specific algorithms, enabling detection of minute tissue displacements that correspond to myocardial deformation [25,36,38]. The transition from one-dimensional tissue Doppler-based strain measurements to two-dimensional speckle tracking has dramatically improved accuracy by accounting for complex cardiac motion patterns including translation, rotation, and through-plane movement [36,38].

Strain parameters quantify myocardial deformation relative to the original tissue dimensions, typically expressed as percentage change from baseline length or area [10,25,38]. Longitudinal strain measures shortening along the long axis of the left ventricle, circumferential strain quantifies circumferential shortening around the ventricular circumference, and radial strain reflects thickening in the radial direction [38]. Each strain component provides unique insights into different aspects of myocardial function and pathology [38].

Advanced strain parameters including global longitudinal strain (GLS), strain rate, and myocardial work indices have demonstrated particular clinical utility for cardiovascular risk assessment [11–24,39]. GLS represents the average longitudinal deformation across all left ventricular segments and has emerged as the most robust and reproducible strain parameter for clinical use [38]. Strain rate parameters provide additional information about the velocity of myocardial deformation and have particular relevance for diastolic function assessment [38].

© 2025 by the author(s). Distributed under a Creative Commons CC BY license.

Preprints.org (www.preprints.org) | NOT PEER-REVIEWED | Posted: 12 February 2026 doi:10.20944/preprints202602.0976.v1

Standardization and Reproducibility

The development of consensus guidelines by the European Association of Cardiovascular Imaging (EACVI) and American Society of Echocardiography (ASE) has been crucial for improving the reliability and clinical applicability of STE when performed according to standardized protocols [9]. These guidelines provide detailed recommendations for image acquisition, analysis protocols, and quality control measures that ensure consistent results across different operators, laboratories, and vendor platforms [9].

Image acquisition standardization encompasses multiple technical considerations including frame rate optimization (50-80 fps for optimal tracking), depth and sector width adjustment to maximize spatial resolution, and gain settings that optimize speckle definition without introducing artifacts [9,10,40]. The guidelines emphasize the importance of obtaining high-quality images with clear endocardial definition, minimal artifacts, and complete myocardial visualization throughout the cardiac cycle [9,40].

Inter-vendor variability represents an ongoing challenge in STE implementation, with different manufacturers employing distinct tracking algorithms and reference standards that can produce systematic differences in strain measurements [25]. While absolute strain values may vary between vendors, the clinical utility of strain measurements for detecting abnormalities and tracking changes over time remains robust across platforms when appropriate vendor-specific reference ranges are applied [25].

Quality control measures include assessment of tracking quality indicators, manual correction of endocardial contours when necessary, and exclusion of segments with poor tracking quality from global strain calculations [9]. The guidelines recommend that global strain values should only be reported when adequate tracking quality is achieved in at least 12 of 16 left ventricular segments, ensuring that calculated values accurately represent true myocardial function [9].

Clinical Validation and Prognostic Value

Extensive clinical validation studies have consistently demonstrated the superior prognostic value of strain parameters compared to conventional echocardiographic measurements [11–24,39]. GLS has shown particular strength as a predictor of cardiovascular events, with meta-analyses demonstrating significant prognostic value across diverse patient populations including those with preserved ejection fraction, heart failure, and ischemic heart disease [11–24,39].

Comparison with other advanced imaging modalities including cardiac magnetic resonance (CMR) has demonstrated that while CMR remains the gold standard for myocardial tissue characterization, STE provides clinically comparable sensitivity for detecting regional dysfunction, with greater practicality for serial assessment in routine clinical settings [36].

The prognostic superiority of GLS over ejection fraction reflects its ability to detect subclinical dysfunction before chamber remodeling and obvious wall motion abnormalities develop [11–24,36]. This enhanced sensitivity is particularly relevant in patients with metabolic disorders and increased visceral adiposity, where early detection of myocardial dysfunction can guide preventive interventions before irreversible changes occur [24,36].

4. Clinical Evidence: VAT and EAT Relationships with Strain

EAT and Left Ventricular Strain Relationships

Comprehensive investigations of the relationship between epicardial adipose tissue and left ventricular strain parameters have consistently demonstrated significant inverse associations across diverse patient populations [3,7,8,41]. A seminal study by Ng et al. examined 130 patients without obstructive coronary artery disease undergoing cardiac computed tomography and three-dimensional speckle-tracking echocardiography, demonstrating that EAT volume was the strongest

© 2025 by the author(s). Distributed under a Creative Commons CC BY license.

Preprints.org (www.preprints.org) | NOT PEER-REVIEWED | Posted: 12 February 2026 doi:10.20944/preprints202602.0976.v1

independent predictor of impaired global longitudinal strain (standardized $\beta = 0.512$, $p<0.001$), even after adjustment for body mass index, waist/hip ratio, and left ventricular volumes [3].

The relationship between EAT and multidirectional strain components reveals differential effects on various aspects of myocardial mechanics [3,7]. While longitudinal strain shows the strongest associations with EAT burden, circumferential and radial strain components also demonstrate significant correlations [3]. This pattern suggests that EAT-mediated myocardial dysfunction affects longitudinally oriented subendocardial fibers most prominently, consistent with the anatomical distribution of EAT and its preferential inflammatory effects on subendocardial regions [3].

Advanced strain parameters including myocardial work indices provide additional insights into the mechanisms linking EAT to ventricular dysfunction. Studies have demonstrated associations between increased EAT burden and impaired myocardial work efficiency in patients with metabolic syndrome [7,41].

The relationship between EAT and strain parameters appears to be independent of traditional cardiovascular risk factors, body mass index, and even overall visceral adiposity [3,4,42,43]. Multivariate analyses consistently demonstrate that EAT volume or thickness remained the strongest predictor of GLS impairment even after adjustment for age, gender, diabetes, hypertension, and BMI, suggesting that anatomical proximity rather than systemic effects primarily drives the EAT-strain relationship [3,42,43].

Temporal dynamics of EAT-strain relationships reveal that strain impairment occurs early in the course of EAT expansion, often preceding changes in conventional echocardiographic parameters [8,41,44]. In the PREDIMAR study, patients with EAT ≥135g demonstrated significantly higher odds of persistent atrial fibrillation [23], suggesting potential thresholds for clinical risk stratification.

VAT and Myocardial Function Relationships

The relationship between visceral adipose tissue and myocardial strain involves more complex systemic mechanisms compared to the predominantly local effects of EAT [27]. Martinez-Dominguez et al. conducted a sophisticated mediation analysis in 150 patients with type 2 diabetes and demonstrated that VAT mediates approximately 60.9% of the relationship between insulin resistance and left ventricular global longitudinal strain [27].

This mediation effect suggests that VAT serves as a critical intermediate pathway linking metabolic dysfunction to cardiac performance, operating through systemic inflammatory cascades, altered substrate metabolism, and neurohormonal activation [27]. The strong mediation percentage indicates that targeting VAT reduction may be particularly effective for improving cardiac function in patients with metabolic disorders [27].

Increased visceral and epicardial adiposity may contribute to heterogeneous myocardial remodeling patterns, though the specific relationships with regional strain distribution require further investigation. The relationship between adipose tissue and myocardial energetic efficiency represents a particularly important mechanistic pathway. Reduced myocardial mechano-energetic efficiency, assessed by stroke volume/heart rate ratio, has been associated with impaired longitudinal and circumferential strain in patients with increased body mass index [45,46]. In metabolic syndrome, epicardial adipose tissue volume negatively correlates with myocardial energetic efficiency, which partially mediates the observed relationship between EAT and impaired global longitudinal strain [41].

Population-based studies including the Framingham Heart Study have provided important insights into the long-term cardiovascular consequences of adipose tissue burden [47,48]. In the Framingham cohort, increased EAT thickness was associated with significantly elevated risk of incident heart failure (HR 1.43, 95% CI 1.19-1.72), with this relationship partially mediated by impaired global longitudinal strain and elevated NT-proBNP levels [47].

© 2025 by the author(s). Distributed under a Creative Commons CC BY license.

Preprints.org (www.preprints.org) | NOT PEER-REVIEWED | Posted: 12 February 2026
doi:10.20944/preprints202602.0976.v1

Atrial Strain and Adipose Tissue Relationships

Left atrial strain parameters demonstrate particularly strong associations with both VAT and EAT, reflecting the intimate anatomical relationships between atrial tissue and surrounding adipose depots [26,49]. The PREDIMAR study, a landmark investigation of Mediterranean diet effects on atrial fibrillation, demonstrated that patients with EAT ≥135g exhibit significantly impaired left atrial strain parameters compared to those with lower EAT burdens [23].

The pathophysiological basis for atrial strain-adipose tissue relationships involves several interconnected mechanisms including direct inflammatory effects on atrial myocardium, promotion of atrial fibrosis through pro-fibrotic mediator release, and alteration of atrial electrophysiological properties [31,50]. EAT surrounding the left atrium demonstrates particularly intense inflammatory activity in patients with atrial fibrillation, creating local environments that promote arrhythmia initiation and perpetuation [31,50].

Atrial strain parameters serve as sensitive markers of atrial myopathy that precede the development of clinically apparent atrial fibrillation [26,49,51]. Studies utilizing comprehensive atrial strain analysis demonstrate that patients with increased EAT exhibit reduced atrial reservoir function, impaired conduit function, and enhanced booster pump function as compensatory responses [26,49,51].

The relationship between visceral adiposity and atrial strain extends beyond structural changes to include metabolic and autonomic effects [31,52]. Increased VAT is associated with enhanced sympathetic activity, altered atrial electrophysiological properties, and increased susceptibility to triggered activity, all of which contribute to atrial strain abnormalities and arrhythmia risk [31,52].

Therapeutic implications of atrial strain-adipose tissue relationships are particularly relevant for atrial fibrillation prevention and management [23]. Studies demonstrating that dietary interventions can simultaneously reduce EAT burden and improve atrial strain parameters suggest that nutritional interventions may favorably modulate epicardial adipose tissue and atrial functional parameters in selected high-risk populations [23].

5. Nutritional Interventions and Strain Outcomes

Mediterranean Diet Evidence

The Mediterranean diet represents the most extensively studied dietary pattern for cardiovascular prevention, with particular relevance for patients with increased visceral and epicardial adiposity [23,53,54]. The PREDIMAR (Prevention of Atrial Fibrillation with Mediterranean Diet) trial demonstrated that Mediterranean diet adherence is associated with lower EAT burden in patients with atrial fibrillation. In a substudy of 199 patients with persistent atrial fibrillation undergoing catheter ablation, higher Mediterranean diet adherence (MEDAS score ≥7) was significantly associated with lower odds of having elevated EAT (≥135 g) compared to lower adherence patterns (multivariable OR 0.45, 95% CI 0.22-0.91, $p = 0.025$) [23]. This inverse association remained statistically significant after adjusting for multiple confounding factors. In addition, a higher EAT burden was significantly associated with persistent atrial fibrillation at baseline (multivariable OR 2.22, 95% CI 1.03-4.79, $p=0.042$) [23].

The mechanistic basis for Mediterranean diet effects on adipose tissue includes anti-inflammatory properties of polyphenols and monounsaturated fats, particularly from extra virgin olive oil and nuts [53–55]. Extra virgin olive oil contains bioactive compounds including oleocanthal and hydroxytyrosol that demonstrate direct anti-inflammatory properties and may specifically target adipose tissue inflammation [54–56]. The PREDIMED trial demonstrated that Mediterranean diet supplemented with extra virgin olive oil or nuts significantly reduced cardiovascular events, supporting the cardiovascular benefits of these specific dietary components [54].

Long-term follow-up data from Mediterranean diet intervention studies reveal sustained effects on both adipose tissue characteristics and cardiac function parameters [23,53]. The protective effects

© 2025 by the author(s). Distributed under a Creative Commons CC BY license.

appear to be maintained for at least 24 months after intervention initiation, suggesting that dietary pattern changes can produce lasting modifications in cardiovascular risk profiles [23,53].

Weight Loss Interventions

Lifestyle modification programs incorporating caloric restriction and exercise training produce substantial improvements in both adipose tissue distribution and strain parameters, with effects often exceeding those achievable through dietary changes alone [57,58]. Meta-analyses of weight loss interventions demonstrated reductions in EAT of 14-31% following intensive lifestyle modifications, with corresponding improvements in global longitudinal strain [58,59]. Prospective studies have shown that each 1 mm reduction in EAT thickness was independently associated with a 0.48% improvement in GLS [59].

The temporal sequence of changes following weight loss interventions reveals that strain improvements often precede detectable changes in conventional echocardiographic parameters [57,60]. Serial strain assessments demonstrate significant improvements as early as 3-6 months after intervention initiation, while changes in ejection fraction and chamber dimensions may not become apparent until 12-18 months, supporting the utility of strain imaging for monitoring early treatment responses [60].

Exercise training effects on strain parameters appear to be independent of weight loss magnitude, suggesting that physical activity provides direct cardioprotective benefits beyond those attributable to adipose tissue reduction [57,61]. Aerobic exercise training specifically improves myocardial energetic efficiency, enhances diastolic function, and reduces systemic inflammation, all of which contribute to strain parameter improvements [57,61].

Combined aerobic and resistance training programs demonstrate superior effects on strain parameters compared to either modality alone, with particular benefits for patients with metabolic syndrome [62]. The optimal exercise prescription includes moderate-intensity aerobic exercise (150 minutes per week) combined with resistance training (2-3 sessions per week), consistent with current cardiovascular prevention guidelines [62].

Bariatric Surgery Outcomes

Bariatric surgery produces dramatic and sustained reductions in both VAT and EAT that significantly exceed those achievable through lifestyle modifications alone. A comprehensive systematic review and meta-analysis by Pereira et al. analyzing 35 studies demonstrated consistent reductions in EAT following bariatric surgery, with 21 of 22 studies showing statistically significant decreases [63]. The pooled effect size for EAT reduction was substantial (ES = -0.89, 95% CI: -1.23 to -0.55, p<0.001), establishing bariatric surgery as the most effective intervention for epicardial fat reduction [63].

The temporal dynamics of EAT reduction following bariatric surgery reveal that maximal benefits occur within the first 6 months post-procedure, with 43% reduction in VAT and 14% reduction in EAT observed within 212 days [63]. Serial imaging studies demonstrate corresponding improvements in global longitudinal strain parameters [63,64].

The mechanisms underlying bariatric surgery effects on strain parameters involve both weight-dependent and weight-independent pathways [63,65]. While substantial weight loss contributes to improved cardiac mechanics through reduced preload and afterload, additional benefits appear to result from altered gastrointestinal hormone profiles, improved insulin sensitivity, and reduced systemic inflammation [65].

Differential effects of various bariatric procedures on cardiovascular outcomes suggest that procedures involving intestinal bypass (Roux-en-Y gastric bypass) may provide superior cardioprotective benefits compared to purely restrictive procedures (sleeve gastrectomy) [66]. Studies have demonstrated lower rates of major adverse cardiovascular events (MACE) with RYGB, with one cohort study finding RYGB was associated with a 25% lower MACE risk (HR 0.75) compared to sleeve gastrectomy over up to 11 years of follow-up [66].

© 2025 by the author(s). Distributed under a Creative Commons CC BY license.

Preprints.org (www.preprints.org) | NOT PEER-REVIEWED | Posted: 12 February 2026 doi:10.20944/preprints202602.0976.v1

Long-term sustainability of bariatric surgery effects on strain parameters appears to be excellent, with maintained improvements demonstrated at 5-year follow-up [67]. However, patients require careful monitoring for potential nutritional deficiencies that could adversely affect myocardial function, particularly deficiencies in thiamine, B12, and essential fatty acids [67].

Emerging Nutritional and Therapeutic Approaches

Plant-based dietary patterns represent an increasingly studied approach for cardiovascular prevention, with preliminary evidence suggesting benefits for both adipose tissue biology and strain parameters [53,68]. Studies of strict plant-based diets demonstrate reductions in systemic inflammation markers, improved endothelial function, and favorable changes in gut microbiome composition that may contribute to cardiovascular protection [68].

Intermittent fasting protocols, particularly time-restricted eating approaches, show promise for targeting visceral adiposity reduction while preserving lean body mass [69]. Preliminary studies suggest that intermittent fasting protocols, particularly when combined with protein pacing, may produce preferential VAT reduction compared to continuous caloric restriction, though effects on strain parameters require further investigation [69].

Specific nutritional components including omega-3 fatty acids, polyphenols, and soluble fiber demonstrate independent effects on cardiovascular function that extend beyond their contributions to weight management [53,66]. Omega-3 fatty acids have been associated with cardiovascular risk reduction and anti-inflammatory effects, primarily through improvements in lipid profiles and systemic inflammatory pathways, although direct effects on cardiac diastolic function and myocardial strain parameters have not been consistently demonstrated [66].

The gut microbiome represents an emerging therapeutic target for modulating the adipose tissue-cardiovascular axis [68]. Probiotic interventions and prebiotic fiber supplementation demonstrate potential for reducing systemic inflammation, improving metabolic function, and enhancing cardiovascular outcomes, though direct effects on strain parameters require additional investigation [68].

Beyond dietary modulation of gut-derived metabolic pathways, pharmacological approaches targeting incretin hormones have shown promising cardiovascular effects. Glucagon-like peptide-1 receptor agonists (GLP-1 RA) demonstrate cardioprotective benefits independently of glycemic control. Meta-analytic evidence shows improvements in diastolic function (E/e' ratio reduction) and favorable left ventricular remodeling (reduced left ventricular mass and left atrial volume) in patients with metabolic disorders, though without significant effects on global longitudinal strain [70]. Preliminary pilot data from dual GLP-1/glucagon receptor agonist studies suggest potential improvements in diastolic strain rate parameters [71]. An overview of key studies is presented in Table 1.

Table 1. Summary of Key Studies on Adipose Tissue and Myocardial Strain.

Study (Year)	Population	Imaging Modality	Key Findings
Ng et al. (2016) [3]	130 patients without obstructive CAD	CT + 3D STE	EAT volume strongest predictor of impaired 3D GLS (β=0.512, p<0.001), circumferential, radial, and area strain; independent of BMI and waist/hip ratio
Martinez-Dominguez et al. (2025) [27]	195 T2D patients	Echo + BIA	VAT mediates 60.9% of IR-GLS relationship; positive VAT-GLS association (β=0.482, p=0.039)
Choy et al. (2023) [47]	1,554 participants, Framingham Heart Study	CMR	EAT thickness associated with incident HF (HR 1.43, 95% CI 1.18-1.73); NT-proBNP and GLS mediates EAT-HF relationship
Barrio-Lopez et al. (2024) [23]	199 AF patients (PREDIMAR)	CT	Higher MedDiet adherence associated with lower EAT (mOR=0.45, 95% CI 0.22-0.91); EAT ≥135g associated with persistent AF

© 2025 by the author(s). Distributed under a Creative Commons CC BY license.

Preprints.org (www.preprints.org) | NOT PEER-REVIEWED | Posted: 12 February 2026 doi:10.20944/preprints202602.0976.v1

Chong et al. (2023) [39]	19,709 patients (meta-analysis)	CT + Echo	EAT associated with cardiac death (OR 2.53), MI (OR 2.63), AF (OR 4.04)
Sun et al. (2023) [7]	194 patients with suspected MetS	Echo	Increased EAT thickness was associated with alterations in myocardial work indices, including reduced global work efficiency, and with worse LV mechanical performance compared with subjects with thinner EAT
Nerlekar et al. (2018) [44]	22 studies (meta-analysis)	Various	EAT associated with diastolic dysfunction (WMD 24.43 mL); inverse correlation with LVEF inconsistent
Abed et al. (2015) [57]	87 AF patients	CMR	Weight management reduced pericardial fat (140.9 to 118.8 cm^3) and LA volumes
Pereira et al. (2023) [63]	35 studies (meta-analysis)	Various	Bariatric surgery: significant EAT reduction; ES=-0.89, 95% CI: -1.23 to -0.55
Lobeek et al. (2024) [26]	82 HFmrEF/HFpEF patients	CMR	EAT independently associated with LA mechanical dysfunction (OR 2.85, 95% CI 1.04-7.79); 41% had LA dysfunction
Wang et al. (2025) [49]	113 HFpEF + 48 controls	Echo	EAT thickness greater in HFpEF (8.0±1.0 vs 5.0±0.7 mm); inverse correlation with LA strain parameters (LASr, LAScd, LASct)

Abbreviations: *AF, atrial fibrillation; BIA, bioelectrical impedance analysis; CMR, cardiac magnetic resonance; CT, computed tomography; EAT, epicardial adipose tissue; Echo, echocardiography; GLS, global longitudinal strain; HF, heart failure; HFpEF, heart failure with preserved ejection fraction; HFmrEF, heart failure with mildly reduced ejection fraction; HR, hazard ratio; IR, insulin resistance; LA, left atrial; LASr, left atrial strain reservoir; LAScd, left atrial strain conduit; LASct, left atrial strain contraction; MedDiet, Mediterranean diet; MetS, metabolic syndrome; MI, myocardial infarction; mOR, multivariable odds ratio; OR, odds ratio; STE, speckle-tracking echocardiography; T2DM, type 2 diabetes; WMD, weighted mean difference.*

6. Clinical Applications and Future Directions

Risk Stratification Strategies

Evidence from prognostic studies suggests that the integration of adipose tissue imaging and myocardial strain assessment may improve cardiovascular risk stratification beyond traditional risk factors [47,48,72].

Target populations for comprehensive imaging-based risk assessment include patients with metabolic syndrome, pre-diabetes, and obesity, particularly those with intermediate risk profiles where traditional algorithms provide limited guidance [27,47,72]. The combination of strain imaging and adipose tissue quantification appears particularly valuable in these populations, where subclinical dysfunction may be present despite normal conventional imaging findings [27,47].

Workflow integration represents a critical practical consideration for implementing combined adipose tissue and strain assessment in routine clinical practice. Contemporary imaging protocols can efficiently incorporate both assessments within standard echocardiographic examinations, with minimal additional time requirements when appropriate software tools and operator training are available.

Emerging Technologies and Research Directions

Artificial intelligence applications represent a promising avenue for advancing adipose tissue and strain assessment, particularly for automated analysis. Machine learning algorithms demonstrate remarkable accuracy for automated EAT quantification, strain analysis, and integrated risk prediction, with potential for real-time clinical decision support and improved measurement reproducibility.

Three-dimensional and four-dimensional strain imaging technologies provide increasingly sophisticated assessments of myocardial mechanics that may reveal subtle abnormalities not detected by conventional two-dimensional approaches [36]. These advanced techniques appear particularly

© 2025 by the author(s). Distributed under a Creative Commons CC BY license.

Preprints.org (www.preprints.org) | NOT PEER-REVIEWED | Posted: 12 February 2026 doi:10.20944/preprints202602.0976.v1

valuable for detecting regional dysfunction patterns associated with specific adipose tissue distributions [36].

Portable and point-of-care echocardiographic technologies are rapidly advancing, with potential for bringing sophisticated strain and adipose tissue assessment capabilities to diverse clinical settings including primary care, remote monitoring, and resource-limited environments. These technological advances may democratize access to advanced cardiovascular imaging and enable population-level screening programs.

Implementation Science and Clinical Integration

Healthcare delivery model considerations are essential for successful integration of advanced imaging approaches into routine cardiovascular care [47]. The development of standardized protocols, training programs, and quality assurance measures will be critical for ensuring consistent implementation across diverse healthcare settings [47].

Population health strategies incorporating strain imaging and adipose tissue assessment may provide powerful tools for primary cardiovascular prevention [47,48]. Community-based screening programs targeting high-risk populations could identify individuals with subclinical dysfunction who would benefit from intensive preventive interventions [47].

Health economics research will be crucial for demonstrating the value proposition of integrated imaging approaches and securing sustainable reimbursement for advanced cardiovascular risk assessment. Studies demonstrating improved clinical outcomes, reduced healthcare utilization, and enhanced quality of life will be essential for supporting widespread adoption.

Global health applications represent important considerations for ensuring equitable access to advanced cardiovascular imaging capabilities, thus supporting the development of cost-effective, portable technologies and simplified protocols may enable implementation in resource-limited settings where cardiovascular disease burden continues to increase.

7. Conclusions

This narrative review summarizes consistent evidence supporting associations between visceral and epicardial adipose tissue burden and impaired myocardial strain parameters. The consistent findings across diverse populations, imaging modalities, and clinical settings support the biological and clinical significance of these relationships for cardiovascular risk assessment and management.

Key findings include inverse associations between adipose tissue burden and global longitudinal strain, with correlation strengths varying across studies and populations. These associations remain significant after adjustment for traditional cardiovascular risk factors, suggesting that adipose tissue distribution provides independent prognostic information that complements established risk assessment approaches.

Proposed mechanistic pathways linking adipose tissue dysfunction to strain abnormalities involve complex interactions between inflammatory activation, metabolic dysfunction, and direct tissue effects that collectively impair myocardial mechanics before conventional abnormalities become apparent. These pathways create opportunities for early intervention through targeted therapeutic approaches that address both adipose tissue biology and cardiovascular function.

Nutritional interventions, particularly Mediterranean diet adherence, demonstrate significant potential for reducing adipose tissue burden. The PREDIMAR trial demonstrated that higher Mediterranean diet adherence is significantly associated with lower epicardial adipose tissue burden in patients with atrial fibrillation (multivariable OR 0.45 for EAT ≥135g), with higher EAT burden significantly associated with persistent atrial fibrillation [23]. These findings, together with emerging evidence on pharmacological modulation of myocardial metabolism and cardiac function [70,71], support the integration of comprehensive therapeutic approaches into cardiovascular prevention strategies.

The superior prognostic value of strain parameters compared to conventional imaging measurements, combined with the modifiable nature of adipose tissue through lifestyle

© 2025 by the author(s). Distributed under a Creative Commons CC BY license.

Preprints.org (www.preprints.org) | NOT PEER-REVIEWED | Posted: 12 February 2026
doi:10.20944/preprints202602.0976.v1

interventions, creates compelling opportunities for personalized cardiovascular prevention. Future research priorities should include large-scale intervention trials with strain endpoints, development of integrated risk prediction algorithms, and implementation science studies to guide clinical adoption.

The convergence of advanced cardiac imaging, metabolic phenotyping, and evidence-based nutritional interventions represents a paradigm shift toward precision cardiovascular medicine that addresses individual risk profiles through mechanistically targeted therapeutic approaches. This integrated approach holds particular promise for addressing the growing global burden of metabolically mediated cardiovascular disease and may ultimately transform primary prevention strategies for high-risk populations.

Author Contributions: SR conceived, wrote and proof-read the manuscript. MV researched, wrote the manuscript, and conceptualized the visualization. AF, CF, and LDC contributed to the revision of the manuscript.

Funding: This research received no external funding.

Data availability statement: No new data were created or analyzed in this study. Data sharing is not applicable to this article.

Conflicts of Interest: The author declares no conflicts of interest.

References

1. Iacobellis G. Epicardial adipose tissue in contemporary cardiology. Nat Rev Cardiol. 2022;19(9):593-606. doi:10.1038/s41569-022-00679-9
2. Iacobellis G, Corradi D, Sharma AM. Epicardial adipose tissue: anatomic, biomolecular and clinical relationships with the heart. Nat Clin Pract Cardiovasc Med. 2005;2(10):536-43. doi:10.1038/ncpcardio0319
3. Ng ACT, Goo SY, Roche N, et al. Epicardial adipose tissue volume and left ventricular myocardial function using 3-dimensional speckle tracking echocardiography. Can J Cardiol. 2016;32(12):1485-1492. doi:10.1016/j.cjca.2016.06.009
4. Pugliese NR, Paneni F, Mazzola M, et al. Impact of epicardial adipose tissue on cardiovascular haemodynamics, metabolic profile, and prognosis in heart failure. Eur J Heart Fail. 2021;23(11):1858-1871. doi:10.1002/ejhf.2337
5. Neeland IJ, Ross R, Després JP, et al. Visceral and Ectopic Fat, Atherosclerosis, and Cardiometabolic Disease: A Position Statement. Lancet Diabetes Endocrinol. 2019;7(9):715-725. doi:10.1016/S2213-8587(19)30084-1
6. Goldman SA, Requena-Ibanez JA, Devesa A, et al. Uncovering the Role of Epicardial Adipose Tissue in Heart Failure With Preserved Ejection Fraction. JACC Adv. 2023;2(9):100657. doi:10.1016/j.jacadv.2023.100657
7. Sun LJ, Xiao CW, Zhao XB, Guo S, Zhang F. Association between epicardial adipose tissue and myocardial work by non-invasive left ventricular pressure-strain loop in people with suspected metabolic syndrome. Sci Rep. 2023;13(1):14415. doi:10.1038/s41598-023-41779-x
8. Geers J, Manral N, Razipour A, et al. Epicardial adipose tissue, myocardial remodelling and adverse outcomes in asymptomatic aortic stenosis: a post hoc analysis of a randomised controlled trial. Heart. 2025;111(14):686-694. doi:10.1136/heartjnl-2024-324925
9. Voigt JU, Pedrizzetti G, Lysyansky P, et al. Definitions for a common standard for 2D speckle tracking echocardiography: consensus document of the EACVI/ASE/Industry Task Force to standardize deformation imaging. Eur Heart J Cardiovasc Imaging. 2015;16(1):1-11. doi:10.1093/ehjci/jeu184
10. Lang RM, Badano LP, Mor-Avi V, et al. Recommendations for cardiac chamber quantification by echocardiography in adults: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. Eur Heart J Cardiovasc Imaging. 2015;16(3):233-70. doi:10.1093/ehjci/jev014

© 2025 by the author(s). Distributed under a Creative Commons CC BY license.

Preprints.org (www.preprints.org) | NOT PEER-REVIEWED | Posted: 12 February 2026
doi:10.20944/preprints202602.0976.v1

11. Sartorio A, Cristin L, Pont CD, Farzaneh-Far A, Romano S. Global longitudinal strain as an early marker of cardiac damage after cardiotoxic medications, a state-of-the-art review. Prog Cardiovasc Dis. 2025;89:92-101. doi:10.1016/j.pcad.2025.01.001
12. Romano S, Kitkungvan D, Nguyen DT, et al. Implications of myocardial strain in primary mitral regurgitation-a cardiovascular magnetic resonance study. Eur Heart J Cardiovasc Imaging. 2024;26(1):126-134. doi:10.1093/ehjci/jeae245
13. Bevilacqua M, De Togni P, Cattazzo F, et al. Global Longitudinal Strain to Predict Respiratory Failure and Death in Patients Admitted for COVID-19-Related Disease. Am J Cardiol. 2022;165:109-115. doi:10.1016/j.amjcard.2021.10.046
14. Romano S, Dell'atti D, Judd RM, et al. Prognostic Value of Feature-Tracking Right Ventricular Longitudinal Strain in Severe Functional Tricuspid Regurgitation: A Multicenter Study. JACC Cardiovasc Imaging. 2021;14(8):1561-1568. doi:10.1016/j.jcmg.2021.02.009
15. Farzaneh-Far A, Romano S. Imaging and Impact of Myocardial Strain in Myocarditis. JACC Cardiovasc Imaging. 2020;13(9):1902-1905. doi:10.1016/j.jcmg.2020.05.028
16. Romano S, Judd RM, Kim RJ, et al. Feature-Tracking Global Longitudinal Strain Predicts Mortality in Patients With Preserved Ejection Fraction: A Multicenter Study. JACC Cardiovasc Imaging. 2020;13(4):940-947. doi:10.1016/j.jcmg.2019.10.004
17. Romano S, Romer B, Evans K, et al. Prognostic Implications of Blunted Feature-Tracking Global Longitudinal Strain During Vasodilator Cardiovascular Magnetic Resonance Stress Imaging. JACC Cardiovasc Imaging. 2020;13(1 Pt 1):58-65. doi:10.1016/j.jcmg.2019.03.002
18. Romano S, Judd RM, Kim RJ, et al. Prognostic Implications of Mitral Annular Plane Systolic Excursion in Patients with Hypertension and a Clinical Indication for Cardiac Magnetic Resonance Imaging: A Multicenter Study. JACC Cardiovasc Imaging. 2019;12(9):1769-1779. doi:10.1016/j.jcmg.2018.10.003
19. Romano S, Sartorio A, Dal Pont C, et al. Subclinical cardiac dysfunction in idiopathic inflammatory myopathies: the role of global longitudinal strain. Clin Exp Med. In Press. doi:10.1007/s10238-026-02054-1
20. Romano S, Judd RM, Kim RJ, et al. Association of Feature-Tracking Cardiac Magnetic Resonance Imaging Left Ventricular Global Longitudinal Strain With All-Cause Mortality in Patients With Reduced Left Ventricular Ejection Fraction. Circulation. 2017;135(23):2313-2315. doi:10.1161/CIRCULATIONAHA.117.027740
21. Romano S, Mansour IN, Kansal M, et al. Left Ventricular global longitudinal strain predicts heart failure readmission in acute decompensated heart failure. Cardiovasc Ultrasound. 2017;15(1):6. doi:10.1186/s12947-017-0098-3
22. Shenoy C, Romano S, Hughes A, et al. Cardiac Magnetic Resonance Feature Tracking Global Longitudinal Strain and Prognosis After Heart Transplantation. JACC Cardiovasc Imaging. 2020;13(9):1934-1942. doi:10.1016/j.jcmg.2020.04.004
23. Barrio-Lopez MT, Ruiz-Canela M, Goni L, et al. Mediterranean diet and epicardial adipose tissue in patients with atrial fibrillation treated with ablation: a substudy of the 'PREDIMAR' trial. Eur J Prev Cardiol. 2024;31(3):348-355. doi:10.1093/eurjpc/zwad355
24. Sartorio A, Dal Pont C, Romano S. Standard and New Echocardiographic Techniques, Such as Global Longitudinal Strain, to Monitor the Impact of Diets on Cardiovascular Diseases and Heart Function. Nutrients. 2024;16(10):1471. doi:10.3390/nu16101471
25. Mirea O, Pagourelias ED, Duchenne J, et al. Intervendor differences in the accuracy of detecting regional functional abnormalities: a report from the EACVI-ASE Strain Standardization Task Force. JACC Cardiovasc Imaging. 2018;11(1):25-34. doi:10.1016/j.jcmg.2017.02.014
26. Lobeek M, Gorter TM, Westenbrink BD, et al. Increased epicardial adipose tissue is associated with left atrial mechanical dysfunction in patients with heart failure with mildly reduced and preserved ejection fraction. Clin Res Cardiol. 2025;114(5):601-608. doi:10.1007/s00392-024-02466-7
27. Martinez-Dominguez P, Gomez-Aviles P, Bautista-García K, et al. Visceral adipose tissue mediates the relationship between left ventricular global longitudinal strain and insulin resistance among adults living with type 2 diabetes. Cardiovasc Diabetol. 2025;24(1):2. doi:10.1186/s12933-024-02547-x

© 2025 by the author(s). Distributed under a Creative Commons CC BY license.

Preprints.org (www.preprints.org) | NOT PEER-REVIEWED | Posted: 12 February 2026
doi:10.20944/preprints202602.0976.v1

28. Mazurek T, Zhang L, Zalewski A, et al. Human epicardial adipose tissue is a source of inflammatory mediators. Circulation. 2003;108(20):2460-6. doi:10.1161/01.CIR.0000099542.57313.BE
29. Packer M. Epicardial adipose tissue may mediate deleterious effects of obesity and inflammation on the myocardium. J Am Coll Cardiol. 2018;71(20):2360-2372. doi:10.1016/j.jacc.2018.03.509
30. Honka H, Solis-Herrera C, Triplitt C, et al. Therapeutic Manipulation of Myocardial Metabolism: JACC State-of-the-Art Review. J Am Coll Cardiol. 2021;77(16):2022-2039. doi:10.1016/j.jacc.2021.02.057
31. Konwerski M, Gąsecka A, Opolski G, Grabowski M, Mazurek T. Role of Epicardial Adipose Tissue in Cardiovascular Diseases: A Review. Biology (Basel). 2022;11(3):355. doi:10.3390/biology11030355
32. Iacobellis G, Bianco AC. Epicardial Adipose Tissue: Emerging Physiological, Pathophysiological and Clinical Features. Trends Endocrinol Metab. 2011;22(11):450-7. doi:10.1016/j.tem.2011.07.003
33. Gaborit B, Venteclef N, Ancel P, et al. Human Epicardial Adipose Tissue Has a Specific Transcriptomic Signature Depending on Its Anatomical Peri-Atrial, Peri-Ventricular, or Peri-Coronary Location. Cardiovasc Res. 2015;108(1):62-73. doi:10.1093/cvr/cvv208
34. Da Dalt L, Cabodevilla AG, Goldberg IJ, Norata GD. Cardiac Lipid Metabolism, Mitochondrial Function, and Heart Failure. Cardiovasc Res. 2023;119(10):1905-1914. doi:10.1093/cvr/cvad100
35. Chen Z, Jin ZX, Cai J, et al. Energy Substrate Metabolism and Oxidative Stress in Metabolic Cardiomyopathy. J Mol Med (Berl). 2022;100(12):1721-1739. doi:10.1007/s00109-022-02269-1
36. Voigt JU, Cvijic M. 2- And 3-Dimensional Myocardial Strain in Cardiac Health and Disease. JACC Cardiovasc Imaging. 2019;12(9):1849-1863. doi:10.1016/j.jcmg.2019.01.044
37. Del Buono MG, Montone RA, Camilli M, et al. Coronary Microvascular Dysfunction Across the Spectrum of Cardiovascular Diseases: JACC State-of-the-Art Review. J Am Coll Cardiol. 2021;78(13):1352-1371. doi:10.1016/j.jacc.2021.07.042
38. Smiseth OA, Rider O, Cvijic M, et al. Myocardial Strain Imaging: Theory, Current Practice, and the Future. JACC Cardiovasc Imaging. 2025;18(3):340-381. doi:10.1016/j.jcmg.2024.07.011
39. Chong B, Chin YH, Jayabaskaran J, et al. Epicardial adipose tissue assessed by computed tomography and echocardiography are associated with adverse cardiovascular outcomes: a systematic review and meta-analysis. Circ Cardiovasc Imaging. 2023;16(5):e015159. doi:10.1161/CIRCIMAGING.122.015159
40. Mor-Avi V, Lang RM, Badano LP, et al. Current and Evolving Echocardiographic Techniques for the Quantitative Evaluation of Cardiac Mechanics: ASE/EAE Consensus Statement. J Am Soc Echocardiogr. 2011;24(3):277-313. doi:10.1016/j.echo.2011.01.015
41. Yan WF, Wang J, Li Y, et al. Epicardial adipose tissue volume is associated with impaired left ventricular strain in patients with metabolic syndrome: the mediating role of myocardial energetic efficiency. Cardiovasc Diabetol. 2025;24(1):325. doi:10.1186/s12933-025-02859-6
42. Ng ACT, Strudwick M, van der Geest RJ, et al. Impact of Epicardial Adipose Tissue, Left Ventricular Myocardial Fat Content, and Interstitial Fibrosis on Myocardial Contractile Function. Circ Cardiovasc Imaging. 2018;11(8):e007372. doi:10.1161/CIRCIMAGING.117.007372
43. Kosmala W, Sanders P, Marwick TH. Subclinical Myocardial Impairment in Metabolic Diseases. JACC Cardiovasc Imaging. 2017;10(6):692-703. doi:10.1016/j.jcmg.2017.04.001
44. Nerlekar N, Muthalaly RG, Wong N, et al. Association of Volumetric Epicardial Adipose Tissue Quantification and Cardiac Structure and Function. J Am Heart Assoc. 2018;7(23):e009975. doi:10.1161/JAHA.118.009975
45. Mancusi C, Midtbø H, De Luca N, et al. Association of myocardial energetic efficiency with circumferential and longitudinal left ventricular myocardial function in subjects with increased body mass index (the FATCOR Study). J Clin Med. 2021;10(8):1581. doi:10.3390/jcm10081581
46. de Simone G, Izzo R, Losi MA, et al. Depressed myocardial energetic efficiency is associated with increased cardiovascular risk in hypertensive left ventricular hypertrophy. J Hypertens. 2016;34(9):1846-1853. doi:10.1097/HJH.0000000000001007
47. Choy M, Yang H, Wang L, et al. Epicardial adipose tissue thickness is associated with incident heart failure and cardiovascular disease risk in the Framingham Heart Study. BMC Med. 2023;21:117. doi:10.1186/s12916-023-02836-4

© 2025 by the author(s). Distributed under a Creative Commons CC BY license.

Preprints.org (www.preprints.org) | NOT PEER-REVIEWED | Posted: 12 February 2026 doi:10.20944/preprints202602.0976.v1

48. Fox CS, Gona P, Hoffmann U, et al. Pericardial fat, intrathoracic fat, and measures of left ventricular structure and function: the Framingham Heart Study. Circulation. 2009;119(12):1586-91. doi:10.1161/CIRCULATIONAHA.108.828970
49. Wang Y, Zhang Q, Chen Y, et al. Unmasking the Epicardial Adipose Tissue-Left Atrial Strain Nexus in HFpEF: A Potential Echocardiographic Signature of Cardiac Adaptation. Echocardiography. 2025;42:e70053. doi:10.1111/echo.70053
50. Wong CX, Ganesan AN, Selvanayagam JB. Epicardial fat and atrial fibrillation: current evidence, potential mechanisms, clinical implications, and future directions. Eur Heart J. 2017;38(17):1294-1302. doi:10.1093/eurheartj/ehw045
51. Thomas L, Marwick TH, Popescu BA, Donal E, Badano LP. Left Atrial Structure and Function, and Left Ventricular Diastolic Dysfunction: JACC State-of-the-Art Review. J Am Coll Cardiol. 2019;73(15):1961-1977. doi:10.1016/j.jacc.2019.01.059
52. Mahajan R, Lau DH, Brooks AG, et al. Electrophysiological, Electroanatomical, and Structural Remodeling of the Atria as Consequences of Sustained Obesity. J Am Coll Cardiol. 2015;66(1):1-11. doi:10.1016/j.jacc.2015.04.058
53. Martínez-González MA, Gea A, Ruiz-Canela M. The Mediterranean diet and cardiovascular health. Circ Res. 2019;124(5):779-798. doi:10.1161/CIRCRESAHA.118.313348
54. Estruch R, Ros E, Salas-Salvadó J, et al. Primary Prevention of Cardiovascular Disease with a Mediterranean Diet Supplemented with Extra-Virgin Olive Oil or Nuts. N Engl J Med. 2018;378(25):e34. doi:10.1056/NEJMoa1800389
55. Schwingshackl L, Morze J, Hoffmann G. Mediterranean Diet and Health Status: Active Ingredients and Pharmacological Mechanisms. Br J Pharmacol. 2020;177(6):1241-1257. doi:10.1111/bph.14778
56. Carpi S, Scoditti E, Massaro M, et al. The Extra-Virgin Olive Oil Polyphenols Oleocanthal and Oleacein Counteract Inflammation-Related Gene and miRNA Expression in Adipocytes. Nutrients. 2019;11(12):E2855. doi:10.3390/nu11122855
57. Abed HS, Nelson AJ, Richardson JD, et al. Impact of weight reduction on pericardial adipose tissue and cardiac structure in patients with atrial fibrillation. Am Heart J. 2015;169(5):655-662.e2. doi:10.1016/j.ahj.2015.02.008
58. Launbo N, Zobel EH, von Scholten BJ, et al. Targeting Epicardial Adipose Tissue With Exercise, Diet, Bariatric Surgery or Pharmaceutical Interventions: A Systematic Review and Meta-Analysis. Obes Rev. 2021;22(1):e13136. doi:10.1111/obr.13136
59. Chin JF, Aga YS, Abou Kamar S, et al. Bariatric Surgery Reduces Epicardial Adipose Tissue and Is Associated With Improved Cardiac Function. Obes Surg. 2025. doi:10.1007/s11695-025-08392-5
60. Baron T, Christersson C, Hjorthén G, et al. Changes in Global Longitudinal Strain and Left Ventricular Ejection Fraction During the First Year After Myocardial Infarction. Eur Heart J Cardiovasc Imaging. 2018;19(10):1165-1173. doi:10.1093/ehjci/jex260
61. Tucker WJ, Fegers-Wustrow I, Halle M, et al. Exercise for Primary and Secondary Prevention of Cardiovascular Disease: JACC Focus Seminar 1/4. J Am Coll Cardiol. 2022;80(11):1091-1106. doi:10.1016/j.jacc.2022.07.004
62. Dor-Haim H, Barak S, Horowitz M, et al. Improvement in Cardiac Dysfunction With a Novel Circuit Training Method Combining Simultaneous Aerobic-Resistance Exercises. PLoS One. 2018;13(1):e0188551. doi:10.1371/journal.pone.0188551
63. Pereira JPS, Calafatti M, Martinino A, et al. Epicardial Adipose Tissue Changes After Bariatric and Metabolic Surgery: a Systematic Review and Meta-analysis. Obes Surg. 2023;33(11):3636-3648. doi:10.1007/s11695-023-06848-0
64. Hammer S, Snel M, Lamb HJ, et al. Prolonged caloric restriction in obese patients with type 2 diabetes mellitus decreases myocardial triglyceride content and improves myocardial function. J Am Coll Cardiol. 2008;52(12):1006-1012. doi:10.1016/j.jacc.2008.04.068
65. Honka H, Solis-Herrera C, Triplitt C, et al. Therapeutic Manipulation of Myocardial Metabolism: JACC State-of-the-Art Review. J Am Coll Cardiol. 2021;77(16):2022-2039. doi:10.1016/j.jacc.2021.02.057

© 2025 by the author(s). Distributed under a Creative Commons CC BY license.

Preprints.org (www.preprints.org) | NOT PEER-REVIEWED | Posted: 12 February 2026

doi:10.20944/preprints202602.0976.v1

66. Wildisen S, Laager R, Struja T, et al. Major Adverse Cardiac Events After Gastric Bypass vs Sleeve Gastrectomy. JAMA Surg. 2025;160(6):690-700. doi:10.1001/jamasurg.2025.1065
67. Snel M, Jonker JT, Hammer S, et al. Long-term beneficial effect of a 16-week very low calorie diet on pericardial fat in obese type 2 diabetes mellitus patients. Obesity (Silver Spring). 2012;20(8):1572-1576. doi:10.1038/oby.2011.390
68. Piché M-E, Tchernof A, Després J-P. Obesity phenotypes, diabetes, and cardiovascular diseases. Circ Res. 2020;126(11):1477-1500. doi:10.1161/CIRCRESAHA.120.316101
69. Arciero PJ, Poe M, Mohr AE, et al. Intermittent Fasting and Protein Pacing Are Superior to Caloric Restriction for Weight and Visceral Fat Loss. Obesity (Silver Spring). 2023;31 Suppl 1:139-149. doi:10.1002/oby.23660
70. Zhang Y, Yang D, Jia Q, Yan J, An F. The Effect of Glucagon-Like Peptide-1 Receptor Agonists on Cardiac Function and Structure in Patients With or Without Type 2 Diabetes Mellitus: An Updated Systematic Review and Meta-Analysis. Diabetes Obes Metab. 2024;26(6):2401-2411. doi:10.1111/dom.15557
71. Goodman J, Schain M, Di Stefano G, et al. Exenatide and glucagon co-infusion increases myocardial glucose uptake and improves markers of diastolic dysfunction in adults with type 2 diabetes. *Sci Rep.* 2025;15(1):21404. doi:10.1038/s41598-025-04559-3
72. Powell-Wiley TM, Poirier P, Burke LE, et al. Obesity and Cardiovascular Disease: A Scientific Statement From the American Heart Association. Circulation. 2021;143(21):e984-e1010. doi:10.1161/CIR.0000000000000973:

Disclaimer/Publisher's Note: The statements, opinions and data contained in all publications are solely those of the individual author(s) and contributor(s) and not of MDPI and/or the editor(s). MDPI and/or the editor(s) disclaim responsibility for any injury to people or property resulting from any ideas, methods, instructions or products referred to in the content.

© 2025 by the author(s). Distributed under a Creative Commons CC BY license.