





Review

The Use of Myocardial Work in Athletes: A Novel Approach to Assess Cardiac Adaptations and Differentiate Physiological Remodeling from Pathology

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Abstract

Myocardial work (MW), derived from non-invasive pressure–strain loop (PSL) analysis, has recently emerged as a promising echocardiographic index for assessing left ventricular performance. It integrates speckle-tracking echocardiography with estimated left ventricular pressure, providing a load-adjusted measure of myocardial performance. This technique addresses the limitations of traditional parameters such as global longitudinal strain (GLS) and ejection fraction (EF), particularly in populations exposed to dynamic loading conditions, such as athletes. Athletic training induces a spectrum of cardiac adaptations, collectively referred to as the “athlete’s heart,” which may mimic or mask pathological conditions. In this context, MW represents a valuable tool to differentiate physiological remodeling from early myocardial dysfunction or underlying cardiovascular disease (e.g., cardiomyopathies, myocarditis). The aim of this review is to explore the physiological rationale for using MW in athletes, evaluate its relationship with performance metrics (e.g., VO_2 max, lactate threshold), and discuss its potential, yet still emerging and not fully validated, role in informing training adaptation and detecting subclinical cardiac conditions. Additionally, we examine MW applications across different sport disciplines (strength, mixed-sport, and endurance), highlighting its role in individualized assessment and risk stratification. By synthesizing current evidence and outlining future research directions, this work emphasizes the potential of MW to become a standard component of cardiovascular evaluation in sports cardiology.

Keywords: myocardial work; athlete’s heart; cardiac remodeling; echocardiography; physiological adaptation; cardiomyopathy; VO_2 max; strain imaging; pressure-strain loop; sports cardiology



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1. Introduction

1.1. Background

The term “athlete’s heart” refers to the morphological, functional, and electrophysiological adaptations in the myocardium that occur due to regular, intense, and prolonged

physical training. These adaptations enable the heart to meet the increased hemodynamic demands of exercise, resulting in enhanced cardiovascular performance [1].

The extent of exercise-induced cardiac remodeling is determined by multiple factors, including training intensity, duration, and frequency. Interindividual variability is further influenced by physiological factors (e.g., sex, age, and baseline fitness), genetic predisposition, and environmental factors such as training conditions and nutrition [2,3].

These adaptations lead to the classification of two primary phenotypes: the endurance-trained athlete's heart and the strength-trained athlete's heart [4].

Endurance athletes (EA), who perform dynamic (isotonic) exercise, typically develop eccentric left ventricular (LV) hypertrophy [5]. This phenotype is characterized by an enlarged LV cavity with a preserved wall thickness-to-chamber diameter ratio. Eccentric hypertrophy results from chronic elevations in cardiac output and reductions in peripheral vascular resistance, typical of aerobic exercise [6]. These changes enhance the heart's ability to deliver oxygenated blood to working muscles during prolonged activity [7]. Functionally, EA show improved lusitropic performance (diastolic relaxation) and contractile reserve, leading to an increased stroke volume (SV) at rest and during exercise [5].

In contrast, power athletes (PA), who perform static (isometric) exercises, demonstrate concentric LV hypertrophy [8]. This adaptation is marked by increased LV wall thickness without significant chamber dilation. Concentric hypertrophy is caused by transient elevations in arterial blood pressure and in peripheral vascular resistance [6]. This remodeling helps the heart to overcome the increased afterload [9]. In PA, the remodeling contributes to a greater contractile force, with a preserved or only slightly elevated stroke volume [1,9].

Mixed sports athletes, such as soccer or basketball players, who involve both dynamic and static exercise, show a hybrid pattern of cardiac remodeling, reflecting the relative contributions of isotonic and isometric training [9].

The electrophysiological profile of the athlete's heart reflects its structural and functional remodeling. Common electrocardiographic (ECG) findings include sinus bradycardia, early repolarization patterns, first-degree atrioventricular (AV) block, Mobitz type I second-degree AV block, and incomplete right bundle branch block [3,10]. Some cardiovascular pathologies commonly observed in athletes can be suspected through ECG, a cost-effective and widely available tool [11].

While the athlete's heart is a physiological adaptation, its structural and electrophysiological features can mimic pathological conditions, posing important diagnostic challenges. For instance, LV hypertrophy in athletes must be distinguished from hypertrophic cardiomyopathy, and conduction abnormalities require differentiation from inherited arrhythmic syndromes. Moreover, transient troponin elevations, while often benign and associated with strenuous exercise, can mimic myocardial injury and pose a diagnostic dilemma [12,13].

Myocardial Work (MW) is an advanced echocardiographic parameter that enables non-invasive evaluation of myocardial function. Unlike traditional metrics such as Global Longitudinal Strain (GLS) and left ventricular ejection fraction (LVEF), MW provides an assessment of myocardial performance that is less sensitive to variations in hemodynamic loading conditions [14].

This advantage stems from its quantification through Pressure-Strain Loops (PSLs), which mirrors the principles of invasive pressure-volume loops (PVLs) [15]. Suga et al. studies showed that the value of MW is directly proportional to intracardiac pressure and contractility. Based on this evidence, Russel et al. developed a non-invasive method to calculate the left ventricular myocardial work (LV MW) [16].

PSLs are obtained by integrating myocardial strain data from speckle-tracking echocardiography with non-invasive estimates of LV pressure, adjusted by brachial cuff measure-

ments and valve timing. The area enclosed by the PSL represents the myocardial work, expressed in mmHg %. It quantifies both global and regional myocardial contractile capacity, offering insights into myocardial energy expenditure and oxygen consumption [17].

The assessment of myocardial function is fundamental for distinguishing physiological cardiac adaptations from structural heart disease and monitoring cardiovascular health.

However, echocardiography has established itself as a valuable diagnostic tool, enabling the identification of various pathological conditions in athletes and the assessment of physiological cardiovascular adaptations resulting from intense physical training [18–20]. MW could improve the diagnostic accuracy of echocardiography in athletes due to its ability to detect subclinical myocardial dysfunction, having a lower dependence on variations in hemodynamic load compared to LV EF and GLS [21].

The aim of this review is to provide a comprehensive overview of MW as a novel tool for the non-invasive evaluation of athletic cardiac performance and the early detection of subclinical myocardial dysfunction. By exploring its mechanisms, clinical relevance, and current evidence, we emphasize the added value of MW indices for myocardial efficiency and adaptability. Special attention is given to the relationship between MW and cardiopulmonary exercise testing (CPET), highlighting its potential to enhance the physiological interpretation of exercise capacity and cardiovascular function in athletes.

1.2. Methods

This review was conducted following established methodologies for narrative literature reviews, with a focus on synthesizing current knowledge regarding the application of MW in athletes. A comprehensive literature search was performed using PubMed, Scopus, and Web of Science. The search strategy aimed to identify clinical trials, cohort studies, observational studies, cross-sectional studies, meta-analyses, consensus statements, and expert guidelines published up to July 2025.

Search terms included combinations of “myocardial work,” “athlete,” “athlete’s heart,” “sports cardiology,” “echocardiography,” “speckle-tracking,” “cardiopulmonary exercise testing,” and “GLS.” Additional articles were identified through manual screening of reference lists from key publications and reviews.

Searches were limited to English-language publications involving human subjects. Studies were included if they examined athletes, sports participants, or individuals undergoing physical training (endurance, strength, or mixed disciplines). Data from the general population were also considered when MW indices or exercise stress echocardiography were discussed in ways that could be extrapolated to athletic populations.

Exclusion criteria comprised editorials, commentaries, letters to the editor, case reports (unless methodologically significant), protocols, and non-peer-reviewed articles. Selection process: Two independent reviewers (A.S. and R.C.) screened titles and abstracts for relevance. Full-text articles were then assessed according to inclusion and exclusion criteria. Discrepancies were resolved by consensus or, if necessary, through consultation with a third reviewer.

2. Myocardial Work: A Different Method to Evaluate Cardiac Function

LVEF is a widely used and accessible echocardiographic measure with recognized strong prognostic value [22]. However, LVEF has several limitations, such as limited sensitivity and marked load dependence, which reduce its accuracy in evaluating myocardial function [23]. Speckle-tracking echocardiography (STE) is a sensitive method for the assessment of cardiac function. Through the measurement of GLS, STE enables the early detection of subclinical LV dysfunction in various cardiac conditions, including valvular disorders, infiltrative diseases, and chemotherapy-related cardiotoxicity [24]. Although

GLS is more accurate than LVEF, it remains influenced by afterload conditions. Consequently, it is not possible to differentiate whether impaired GLS results from reduced myocardial contractility or from increased afterload.

To overcome these limitations, a novel technique, MW, was introduced in 2018. This method integrates GLS analysis with non-invasively estimated LV pressure, providing a more accurate and load-adjusted evaluation of myocardial performance [14]. Table 1 summarizes the main differences between GLS and MW in the assessment of cardiac function and physiological adaptations.

Table 1. Differences between GLS and MW in the assessment of cardiac function and physiological adaptations.

Parameter	Global Longitudinal Strain (GLS)	Myocardial Work (MW)
Principle	Detects myocardial longitudinal deformation (strain) during systole	Quantifies myocardial performance and energy consumption via pressure–strain loops (PSLs).
Methodology	Tracks myocardial speckles to assess LV deformation in the longitudinal direction, expressed as a negative percentage (–%).	Integrates speckle-tracking strain data with estimated LV pressure to calculate myocardial work (mmHg·%).
Parameters	LV global longitudinal strain (GLS).	<ul style="list-style-type: none"> • Global Work Index (GWI) • Global Constructive Work (GCW) • Global Wasted Work (GWW) • Global Work Efficiency (GWE)
Load dependency	Highly preload- and afterload-dependent, limiting reliability under variable hemodynamic conditions.	Less load-dependent, as LV pressure is incorporated into calculations.
Detection of Physiological Adaptations	Interpretation for differentiating physiological remodeling in athletes is limited by load dependency.	Quantifies myocardial efficiency and oxygen consumption. Emerging evidence supports its use in distinguishing physiological adaptations from pathology.
Sensitivity to detect subclinical dysfunctions	Detects subclinical systolic dysfunction earlier than LVEF.	Identifies subclinical dysfunction through GWW and GWE changes, often before GLS or LVEF abnormalities.
Correlation with CPET	Weak correlation between GLS and CPET-derived VO_2/kg due to load dependence.	Moderate correlations between MW indices and CPET-derived VO_2/kg in both semi-recumbent ergometer and treadmill protocols.
Limits	Operator-dependent; strongly influenced by load conditions. Limited capacity to estimate energy expenditure.	Operator-dependent; requires specialized software and trained operators. Reference benchmarks still need to be established.

PVLs, first introduced by Otto Frank in 1895, have become a cornerstone for understanding LV mechanics and cardiac performance [25]. These loops are constructed from invasive measurements obtained in the catheterization lab. They graphically illustrate the interactions between preload, afterload, and LV contractility during the cardiac cycle, which include four main phases: isovolumic contraction, ejection, isovolumic relaxation, and filling [25]. The slope from the start of the curve to peak pressure at the end of LV ejection reflects myocardial contractility, whereas the area of the PVL corresponds to stroke work and is closely associated with myocardial oxygen consumption. Myocardial efficiency, as represented by these loops, remains highly sensitive to changes in loading conditions [26].

The concept of MW was first introduced in 1979 by an experimental study conducted by Suga et al. [27]. In this study, the investigators validated the concept of MW by measuring PVL during cardiac catheterization. They demonstrated that the PVL area reflects MW and myocardial oxygen consumption. However, the invasive assessment of MW is difficult to perform in routine clinical practice. Therefore, Russell et al. subsequently introduced a novel non-invasive method for measuring MW [28].

Their technique combines estimated LV pressure curves with myocardial strain data, aligning peripheral systolic blood pressure (SBP) with cardiac timing events, as determined by echocardiographic identification of mitral and aortic valve opening and closure. This method allows the generation of regional LV pressure-strain loops, which closely correlate with invasively obtained measurements and reliably represent myocardial energy consumption.

MW integrates strain analysis, obtained by automated functional imaging, with estimated LV pressure data. MW assessment is conducted using a proprietary algorithm (GE Healthcare, Pewaukee, WI, USA). Firstly, standard apical 2-, 3-, and 4-chamber views are acquired for GLS analysis via STE. An estimated LV pressure curve is generated by aligning a reference curve to the duration of isovolumic and ejection phases, typically based on the timing of aortic valve closure. Automated functional imaging is used to trace myocardial borders and calculate GLS, with manual adjustment available to optimize contour accuracy. The analysis produces a bull's-eye plot of regional strain, along with calculated GLS values. Subsequently, SBP is measured noninvasively at the time of image acquisition using a brachial cuff [29]. This value is then entered into the echocardiographic software to estimate peak LV systolic pressure and generate MW quantification.

Once SBP is entered, the software generates an MW bull's-eye plot, analogous to GLS. MW is described through four key parameters that enhance the assessment of LV mechanics:

1. Global Work Index (GWI), mmHg %: total work performed by the left ventricle during mechanical systole, including isovolumic contraction and relaxation. It is visually represented by the area within the pressure-strain loop. Normal values were established in the EACVI NORRE study [30] and range between 1310 and 2538 mmHg % in females and 1270–2428 mmHg % in males;
2. Global Constructive Work (GCW), mmHg %: work performed by the left ventricle that contributes to LVEF during systole. It corresponds to positive work (shortening) during systole and negative work (lengthening) during isovolumic relaxation. Normal ranges are 1543–2924 mmHg % in females and 1650–2807 mmHg % in males [30];
3. Global Wasted Work (GWW), mmHg %: work performed by the left ventricle that does not contribute to LVEF during systole. It corresponds to negative work (longitudinal lengthening) during systole plus positive work (shortening) during isovolumetric relaxation. Normal values are 239 ± 39 mmHg % in females and 238 ± 33 mmHg % in males [30];
4. Global Work Efficiency (GWE), %: ratio between constructive work and total (constructive and wasted) work. It is obtained by the ratio of GCW to the sum of GCW and GWW ($GWE = GCW / [GCW + GWW]$, 0–100%). Normal values in healthy controls are 91 ± 1 mmHg % in females and 90 ± 1.6 mmHg % in males [29,30].

Similarly to GLS, color-coded maps allow for quick visualization of regional differences, and MW indices can be assessed globally or by individual segments.

The use of MW has been studied in various clinical settings, such as arterial hypertension, valvular heart diseases, heart failure, coronary artery disease (CAD), and cardio-oncology.

In hypertensive patients, MW is often increased, without significant alterations in LVEF and GLS [31]. Hypertension, as a condition characterized by increased afterload, leads to higher GWI and GCW, without a significant rise in GWW or a reduction in myocardial efficiency [32]. Therefore, the myocardium is not inefficient in the setting of increased afterload, but it compensates by increasing its workload. Moreover, the severity of arterial hypertension is closely related to the increase in GWI [31].

MW has been studied in acute and chronic coronary syndrome. Zhang et al. [33] evaluated the diagnostic utility of MW in patients with CAD. Among 131 individuals presenting with stable chest pain, who underwent MW assessment and coronary angiography. Both GWI and GCW demonstrated good sensitivity and specificity in identifying high-risk stable CAD. Moreover, regional MW assessment across the main coronary territories showed excellent diagnostic accuracy in predicting high-risk disease. In another study, Boe et al. [34] showed that MW was superior to conventional echocardiographic markers, such as LVEF and GLS, in identifying acute coronary occlusion in patients with non-ST-segment elevation myocardial infarction (NSTEMI). The presence of more than four contiguous segments with MW values below 1700 mmHg % yielded a sensitivity of 81% and specificity of 82% for detecting occlusive CAD. They also proposed the use of regional MW for identifying patients who require prompt invasive treatment. Similarly, Jin et al. [35] identified MW as a promising tool for assessing coronary microvascular dysfunction in post-ST-elevation myocardial infarction (STEMI) patients.

The use of MW has also been evaluated in the field of cardio-oncology. The recent European guidelines establish that the diagnosis and classification of cancer therapeutics-related cardiac dysfunction (CTRCD) is based on symptoms combined with LVEF, GLS, and cardiac biomarkers [36]. GLS has proven to be more sensitive than LVEF for detecting CTRCD. However, it is influenced by loading conditions, and differences in SBP between echocardiographic examinations represent a significant limitation for serial comparisons. In a large retrospective study, Zhan et al. investigated LVEF, GLS, and MW parameters before, during, and after anthracycline chemotherapy, and they demonstrated the superiority of MW over GLS in early CTRCD detection. Specifically, a 15% reduction in GWW and GWI preceded CTRCD defined by GLS, and the changes in GWI and GCW showed a direct correlation with the cumulative anthracycline dose administered. The utility of MW indices compared to conventional echocardiographic parameters was also studied in a cohort of children and young adults receiving anthracycline therapy. In this study, Zhan et al. [37] demonstrated that MW provides an earlier and more sensitive marker of progression toward CTRCD than traditional measures, such as LVEF and GLS. Contrary to these findings, Guan et al. [38] found that the sensitivity of GWI and GWE in predicting CTRCD was lower compared with GLS. Specifically, they concluded that GLS is superior to GWI in predicting CTRCD, particularly in patients with minimal blood pressure variability. Similarly, Calvillo-Argüelles studied breast cancer patients receiving anthracycline and trastuzumab therapy, concluding that MW indices did not offer any measurable improvement over GLS and clinical risk factors in predicting CTRCD identified at subsequent visits [39]. Therefore, more studies are needed to determine the clinical value of MW in the field of cardio-oncology and CTRCD.

Recent research has increasingly emphasized the utility of MW indices in the assessment of non-ischemic cardiomyopathies, a crucial step in differentiating pathological remodeling from the physiological adaptations seen in athletes. In hypertrophic cardiomyopathy (HCM), MW analysis has demonstrated impaired global and regional myocardial performance, particularly reduced constructive work and elevated wasted work, reflecting subclinical dysfunction despite preserved ejection fraction [40,41]. Hiemstra et al. further showed that lower GWI and efficiency were independently associated with adverse

outcomes in nonobstructive HCM, while Jacquemyn et al. extended these findings to pediatric populations, identifying MW as a prognostic marker that tracks disease progression over time [42,43]. In dilated cardiomyopathy (DCM), non-invasive MW assessment similarly offers incremental diagnostic and prognostic value. Chan et al. first established that MW could distinguish DCM from hypertensive remodeling, while Cui et al. linked reduced GWI to the extent of myocardial fibrosis, supporting its role as a marker of disease severity [31,44]. Subsequent studies confirmed the prognostic implications of MW: lower GWI was associated with worse outcomes in nonischemic DCM and advanced heart failure [45,46]. Moreover, a recent study demonstrated that MW analysis, specifically GWE and GWW, enables early detection of subclinical dysfunction in genetically predisposed individuals with familial DCM, even before overt systolic impairment occurs [47]. Beyond HCM and DCM, Li et al. recently found that impaired MW across multiple chambers in restrictive cardiomyopathy predicts adverse prognosis, underscoring its broader applicability across phenotypes of myocardial disease [48].

3. Myocardial Work and CPET: Markers of Athletic Performance

CPET is the gold standard for assessing cardiorespiratory fitness in athletes, as it provides an integrated evaluation of cardiovascular, respiratory, and muscular responses during exercise. Unlike traditional ergometric tests, CPET offers detailed physiological insights through direct measurement of parameters such as oxygen consumption (VO_2), carbon dioxide production (VCO_2), and ventilatory efficiency (e.g., VE/VCO_2 slope), which are key indicators of exercise capacity and endurance performance [49,50]. In athletic populations, CPET is essential for tailoring individualized training programs, monitoring adaptation to exercise, and evaluating recovery after injuries or periods of deconditioning [51]. It also plays a crucial role in differentiating physiological adaptations, such as those of the “athlete’s heart,” from pathological conditions such as cardiomyopathies, particularly when combined with advanced echocardiographic techniques [52]. By providing objective data on both maximal (e.g., peak VO_2) and submaximal (e.g., anaerobic threshold) performance metrics, CPET enables precise assessment of functional capacity, supports safe return-to-play decisions, and helps minimize the risk of re-injury or overtraining in competitive and recreational athletes [51,53].

Several studies have explored the relationship between MW parameters, such as GCW, GWW, GWI, and GWE, and key indicators of aerobic performance like maximal oxygen uptake ($\text{VO}_{2\text{max}}$) or the VE/VCO_2 slope, a key submaximal CPET parameter linked to ventilatory efficiency.

In a pediatric context, Zhao et al. evaluated pre-adolescent male basketball players and found that GWI and GWE indexed by body mass correlated significantly with relative $\text{VO}_{2\text{max}}$ ($r = 0.58$ and $r = 0.69$, respectively) and with peak O_2 pulse, underscoring the potential of MW to assess aerobic performance and cardiac adaptation even in young athletes [54].

Tokodi et al. demonstrated in elite swimmers that resting GWI was positively correlated with VO_2/kg measured during CPET ($r = 0.527$, $p < 0.001$), suggesting that greater myocardial contractile efficiency at rest reflects superior aerobic capacity [55]. Notably, athletes in this study exhibited higher resting GWI compared to sedentary controls, despite having lower GLS values, a typical feature of the “athlete’s heart”.

However, when comparing populations with different training statuses, variations emerge. Segreti et al. showed in athletes with knee injuries that MW parameters at rest predicted exercise capacity despite significant deconditioning. Specifically, peak VO_2 was positively correlated with GWE ($r = 0.455$, $p = 0.012$) and negatively with GWW ($r = -0.441$, $p = 0.015$), while the VE/VCO_2 slope correlated inversely with GWE ($r = -0.585$, $p = 0.001$)

and positively with GWW ($r = 0.499$, $p = 0.005$), highlighting the ability of MW to estimate both performance capacity and ventilatory efficiency [56].

Erevik et al. provided compelling data from middle-aged recreational athletes undergoing both CPET and a 91 km cycling race. They found that resting GWW was an independent predictor of performance: higher GWW correlated with longer race duration ($\rho = 0.42$, $p = 0.008$) and lower mean power output during both CPET and competition, indicating that myocardial inefficiency may limit endurance even in otherwise healthy individuals [57]. These findings reinforce the concept that myocardial inefficiency can limit endurance, even in otherwise healthy recreational athletes. The inverse relationship between GWW and aerobic performance suggests that reducing energy loss during systole may be an important component of endurance optimization.

Kandels et al. investigated Δ GWI, the difference between pre- and post-CPET GWI, in competitive athletes, demonstrating moderate correlations between Δ GWI and relative VO_2max in handball ($r = 0.631$) and football players ($r = 0.592$). These results suggest that increases in GWI after maximal exercise are associated with higher aerobic capacity and may reflect contractile reserve [58].

D'Andrea et al. analyzed 350 EA and 150 controls, reporting that GWE at rest was closely related to peak VO_2 , maximal wattage, and pulmonary pressures measured during exercise, positioning it as a predictor of functional capacity even when GLS is reduced at rest [59].

Not all studies, however, report a direct correlation between MW and CPET parameters. Di Gioia et al. reported in 306 Olympic EA that resting GWI and GCW correlated with structural parameters and peak SBP during CPET, but not directly with $\text{VO}_2\text{max}/\text{kg}$, suggesting that in highly trained populations, resting MW may primarily reflect chronic cardiac remodeling rather than current fitness [60]. This may indicate a plateau effect in elite athletes, where high baseline cardiac efficiency masks performance differences, and MW becomes less reflective of functional status. These findings contrast with those of Kandels et al., who reported that Δ GWI (change from pre- to post-exercise) correlated moderately with VO_2max in competitive team sport athletes, supporting the idea that dynamic MW assessments may be more suitable to differentiate aerobic capacity in highly trained individuals [58].

The type of athletic training also appears to modulate the relationship between MW and CPET parameters. D'Andrea et al. examined MW efficiency in professional athletes with physiological left ventricular hypertrophy and demonstrated that, despite the concentric remodeling associated with strength training, GWE remained within normal ranges. These findings support the value of MW as a sensitive tool for distinguishing physiological adaptation from pathological hypertrophy in strength-trained athletes [8].

Additional evidence from Borzi et al. showed that GCW and GWI increased during exercise in both EA and PA, highlighting MW's responsiveness to exercise-induced myocardial demand and suggesting that dynamic MW measurements could complement resting assessments for individualized exercise prescription [61].

Recent evidence also highlights the potential of MW parameters for monitoring changes in exercise capacity during recovery from injury and throughout training cycles. In a recent pilot study by Segreti et al. (2024) involving 22 amateur athletes undergoing knee surgery, resting MW indices were assessed before and after surgery in combination with CPET-derived measures of exercise capacity. The study revealed a significant increase in resting heart rate and improvements in myocardial contractility after surgery, specifically an increase in GWE and a reduction in GWW. However, these improvements in myocardial performance at rest did not correspond to significant changes in peak VO_2 or

VE/VCO₂ slope measured during CPET, suggesting that peripheral factors such as muscle deconditioning may offset central cardiovascular adaptations [56].

These findings underscore the value of MW for tracking cardiac recovery independently of peripheral factors and highlight its potential as a non-invasive marker to monitor functional improvement or persistent limitations following injury. Serial MW measurements could thus provide unique insights into myocardial adaptations over time, supporting individualized return-to-play decisions and optimizing training periodization by distinguishing between central cardiac recovery and ongoing peripheral deconditioning.

Furthermore, combining MW with CPET may enhance the ability to comprehensively evaluate athletes during rehabilitation, detect discrepancies between cardiac and peripheral recovery, and guide multidisciplinary management to restore peak performance while minimizing re-injury risk [51]. Nevertheless, discrepancies across studies highlight the importance of considering context: elite conditioning, structural adaptations, and the balance of central versus peripheral limitations all influence MW–performance relationships.

4. Myocardial Work and Cardiovascular Health in Athletes

Traditional echocardiographic parameters, such as LV wall thickness, cavity size, and EF, are often insufficient to distinguish physiological from pathological remodeling, particularly in highly trained individuals with borderline or heterogeneous changes in systolic function [22,62]. Although GLS provides greater sensitivity for detecting subclinical dysfunction, its strong load dependency limits its accuracy in characterizing physiologic remodeling [63,64].

In this context, MW has emerged as a promising tool that incorporates afterload into the assessment of myocardial function [65,66]. GWI, GCW, GWW, and GWE together provide a more comprehensive profile of myocardial energetics.

In a study on professional football players, Refoyo et al. observed that although these athletes exhibited greater LV volumes and mass, their GCW was significantly lower, and GWI showed a trend toward reduction, indicating a possible adaptive myocardial energy-saving mechanism [21]. During exercise, GWI and GCW increase with systolic deformation, while GWE tends to decline, likely reflecting temporary post-exercise inefficiency rather than pathology [67].

In EA, Huang et al. confirmed that MW parameters adapt dynamically during exercise stress echocardiography, with consistent patterns observed across sexes [67]. D'Andrea et al. also demonstrated that EA have higher resting GWI and GCW compared to detrained peers, despite slightly lower GLS values, highlighting the incremental diagnostic value of MW in athletic cardiac remodeling [59].

Risk stratification in athletes is a growing field, particularly concerning arrhythmogenic substrates and adverse cardiac remodeling. Although atrial enlargement and fibrosis have been implicated in the pathogenesis of atrial fibrillation (AF) in athletes, the ability to identify individuals at highest risk remains limited [68].

Recent evidence suggests that combining MW parameters with advanced atrial imaging may improve risk stratification. Reduced GWE or increased GWW, particularly in the context of impaired LA strain, may identify maladaptive remodeling and early electromechanical dysfunction. LA strain, specifically the reservoir component, is increasingly recognized as an independent marker of atrial function and predictor of arrhythmic risk in athletes [69,70].

Integrating MW and LA strain provides a multidimensional perspective on the cardiac response to training. For example, preserved MW parameters accompanied by mild reductions in LA strain may be consistent with physiological remodeling, whereas concur-

rent impairment in both indices may indicate pathological adaptation, warranting closer follow-up or temporary detraining.

Given the heterogeneity of training modalities, sport disciplines, and athlete phenotypes, the interpretation of MW should be context-specific. Although normative values for MW indices are still being defined, accumulating evidence supports their utility in evaluating the athlete's heart, guiding clinical decision-making, and potentially preventing adverse outcomes.

5. MW in Different Athletic Populations

MW provides a more accurate assessment of LV function because it accounts for afterload, which is often reduced in EA due to chronic peripheral vasodilation induced by training. A study conducted by D'Andrea et al. [59] compared GLS and MW in 350 EA with 150 healthy controls. Although resting GLS was significantly reduced in EA compared with controls (-18.4 ± 2.6 versus $-22.4 \pm 3.3\%$; $p < 0.01$), GWE showed no significant difference, suggesting preserved systolic function despite apparent GLS reduction. Moreover, resting GWE was strongly correlated with exercise performance metrics such as peak VO_2 , maximal watts, and peak E/e' . Regular endurance training was associated with reduced GLS but increased GWI at rest, and GWI demonstrated a positive correlation with VO_2/kg , which is a key indicator of aerobic performance [55]. These findings suggest that MW parameters, especially GWI and GWE, may be more representative of true systolic function in EA than GLS or LVEF. Resting GWI may serve as a potential predictor of cardiac performance in trained individuals. Further prospective studies are needed to establish athlete-specific reference values and to clarify the prognostic role of MW in this population.

The use of MW to evaluate LV function in PA was investigated in a study including thirty-six professional young male athletes engaged in wrestling (the athlete group) and thirty-two healthy age-matched young men (the control group) [71]. This study revealed that after long-term intensive training, the athletes not only showed LV concentric remodeling, but also developed subclinical changes in LV contractility as evidenced by increased GWW and decreased GWE. The best predictor of LV myocardial contractile performance in the athletes was GWE. Therefore, compared with the conventional echocardiographic parameters, the MW appears to be a promising instrument to evaluate progressive changes in LV performance and produce incremental practical significance to detect LV myocardial function abnormalities. D'Andrea et al. [8] evaluated 250 elite PA (e.g., weightlifters, bodybuilders, boxers, rugby players) versus 180 controls. Despite a modest reduction in resting GLS, GWE was maintained and comparable to controls. In the same cohort, resting GWE was the strongest independent predictor of peak watts, VO_2 max, pulmonary artery systolic pressure (PASP) and B-lines at peak exercise (all $p < 0.001$). This demonstrates that GWE not only reflects efficient myocardial function at rest but also correlates with dynamic cardiac performance under stress. The study further highlights that MW is less influenced by afterload than conventional GLS, thereby enhancing its reliability in the pressure-overload physiological conditions typical of strength training. Moreover, while endurance athletes show reduced GLS but increased GWI at rest [55], PA exhibit similar patterns of myocardial adaptation, with concentric remodeling and preserved efficiency. PA also shows a stronger correlation between MWE and PASP, reflecting adaptation to pressure-overload conditions. Further studies are needed to establish reference values and prognostic thresholds for MW parameters in PA. Interestingly, although both endurance and PA demonstrate reduced GLS at rest, their MW profiles differ: EA exhibit elevated GWI, whereas PA maintains normal GWI but shows increased GWW in some cases. These patterns may reflect different types of myocardial adaptation, volume overload versus pressure overload. Moreover, variation in MW parameter thresholds across studies could

stem from differences in strain analysis platforms, blood pressure measurement protocols, or indexing methods (e.g., whether GWI is normalized to body surface area or mass).

Zhao et al. [54] evaluated MW in adolescent male basketball players before and after cardiopulmonary exercise testing. At rest, athletes showed lower GWI and GWE compared to sedentary controls. Under stress, GWE correlated with VO_2 max and oxygen pulse. When indexed by body mass, both GWI and GWE showed strong associations with relative VO_2 max and peak oxygen pulse [72]. In another study, Refoyo et al. [21] assessed investigated MW parameters in elite soccer players: GWI tended to be lower, and GCW was significantly reduced in soccer players vs. controls. GLS and GWE also varied with septal thickness, underscoring load-independent insights provided by MW [21]. These findings suggest that resting MW indices (GWI, GWE) reflect myocardial adaptation patterns specific to mixed training backgrounds (e.g., basketball, football). Exercise-induced MW changes (especially GWI/GWE under stress) correlate with aerobic performance metrics, including CPX-derived VO_2 max and oxygen pulse. MW provides load-adjusted insights superior to GLS, capturing both contractile reserve and physiological remodeling. Therefore, MW shows promise in helping differentiate a normal athlete's heart from early subclinical dysfunction, particularly under stress conditions.

However, when comparing across studies, discrepancies become evident, particularly related to training load and population heterogeneity. For example, Tokodi et al. found increased GWI and preserved GWE in EA, whereas Zhao et al. observed reduced GWI and GWE in pre-adolescent basketball players at rest. These differences likely reflect not only age and developmental stage but also variation in training intensity, chronicity, and timing of evaluation relative to exercise (e.g., immediate post-training vs. true resting state) [54,55]. Overall, while MW parameters offer promise in evaluating athletic cardiac adaptation, inconsistencies across studies highlight the importance of standardized methodologies, population stratification (by age, sex, training volume), and consistent indexing methods.

A summary of key studies evaluating MW indices and their findings in various athlete populations is presented in Table 2.

Table 2. Papers on MW analysis in athletes and findings.

Study	Type of Study and Population	Findings
Refoyo et al., 2023 [21]	Prospective, single-center cohort study 97 people: 49 professional football players and 48 controls. The mean age is 30.48 ± 7.20 years old. The number of males and females is not known.	<ul style="list-style-type: none"> 94.7% of professional players had lower GWW values and 89.5% of them showed higher GWE, compared to normal indices. They have lower GCW and a tendency to show lower GWI values. MW can be helpful in analyzing the physiological remodeling of athletes who have shown better execution capacity and greater contractile reserve during effort.
Tokodi et al., 2022 [55]	Mixed: Animal model + Cross-sectional human study 40 people: 20 elite swimmers (50% males, 50% females) and 20 healthy sedentary controls (50% males, 50% females). The mean age of swimmers was 20 ± 5 years and the control group's mean age was 22 ± 3 years.	<ul style="list-style-type: none"> Swimmers have decreased values of GLS but increased in GWI and GCW, due to elevated resting systolic blood pressure and LV dilation. MW values correlated with invasively measured LV contractility in rats and correlated moderately with CPET-derived VO_2/kg, justifying that supernormal values of GWI and GCW measured during resting conditions indicate better performance during exercise. MW was able to capture the supernormal LV systolic performance of human athletes even during resting conditions.

Table 2. Cont.

Study	Type of Study and Population	Findings
D'Andrea et al., 2020 [59]	Cross-sectional observational study 350 EA: males are 58.5% of the total, females are 41.5%. 150 healthy controls: 85 male (57.4%) and 65 female. The mean age was 31.6 ± 4.2 years.	<ul style="list-style-type: none"> They highlight the possible utility of MW as a good early predictor of physiological remodeling in baseline examination, for cardiac management and decision making in endurance athletes.
Zhao et al., 2022 [54]	Cross-sectional observational study 20 pre-adolescent male basketball players. The mean age was 9.7 ± 1.1 year.	<ul style="list-style-type: none"> GWE at stress correlates with VO_2max, GWI and GWE indexed by body mass correlates with relative VO_2max and GWI and GWE indexed by body mass correlates with oxygen pulse. GWI of pre-adolescent males was lower than normal children. They explain this inconsistency between the current study and previous research by the sensitivity of the GWI to afterload and, hence its wide range of normal values overlapping with pathological ones.
Borzi et al., 2022 [61]	Observational 30 healthy males divided into three groups of 10: sedentary, EA and PA. The mean age was 26.9 ± 6.3 years.	<ul style="list-style-type: none"> In the stress phase, EA and PA show GWI values greater than sedentary, while GWE is reduced in all groups, but higher in EA than in PA. The use of GLS and MW could open new perspectives for the development of personalized diagnostic-therapeutic protocols for the different "types of AH," for the early screening of pathological alterations.
Sengupta et al., 2020 [72]	Cross-sectional observational 24 recreational athletes with a mean age of 41.8 ± 7.4 years. 23 of 24 are males (98%).	<ul style="list-style-type: none"> Through myocardial performance indices, they identified two categories of response to the half-marathon: a group of athletes who completed the half-marathon without a significant change in GWI and a second group who completed the half-marathon by increasing their GWI. This could represent an early manifestation of myocardial stress, an initial increase in myocardial consumption with increased heart rate, which could be a precursor to myocardial fatigue.
D'Andrea et al., 2022 [8]	Cross-sectional with stress testing 250 PA: 155 males (62%) and 95 females. 180 age- and sex-comparable healthy controls. Mean age: 33.6 ± 4.8 years.	<ul style="list-style-type: none"> In PA, there is a stronger relationship between MWE and PASP; in EA, MWE is mainly associated with E/e: these results can be explained by the different loading conditions of the two groups. In PA, MW is related to exercise capacity and left ventricular CR, and could have a potential role in differentiating physiological and pathological conditions.
Da Luz et al., 2025 [73]	Cross-sectional observational 75 professional soccer athletes and 23 recreational athletes, between 18 and 35 years. The number of males and females is not known.	<ul style="list-style-type: none"> The two groups showed no difference in GCW, GWW and GWE, however, GWI was lower in PA. Soccer athletes have normal left ventricular function, while the decrease in GWI is related to cardiac calcium remodeling: lower blood pressure and left ventricular dilation. An important limitation of the study is the assessment of MW only at rest.

Table 2. *Cont.*

Study	Type of Study and Population	Findings
Di Gioia et al., 2025 [60]	Cross-sectional with CPET and stress echo 306 Olympic EA, 170 (55.5%) males. Mean age: 26.3 ± 4.3 years old.	<ul style="list-style-type: none"> MW indices were associated with cardiac remodeling due to their correlation with standard echocardiographic parameters, but not with VO₂ max. It should therefore not be considered as a surrogate marker of an athlete’s training status.
Grandperrin et al., 2023 [74]	Cross-sectional comparative study 24 strength-trained asymptomatic athletes using anabolic androgenic steroids (AAS) (age: 32.3 ± 7.7). 22 athletes diagnosed with HCM (age: 34.8 ± 12.5). 20 healthy control athletes (34.5 ± 7.7). The number of males and females is not known.	<ul style="list-style-type: none"> GWE was significantly diminished in HCM and AAS athletes. GCW and GWE regional analysis showed basal septal segments preferentially affected in AAS-Athletes, and both septal and apical segments affected in HCM-Athletes. This could help clinicians to differentiate between these 2 forms of pathological hypertrophy.

Figure 1 presents a comparison of myocardial work indices (GWI, GCW, GWW, GWE) across sedentary individuals, endurance athletes, power athletes, and subjects with pathological cardiac conditions.

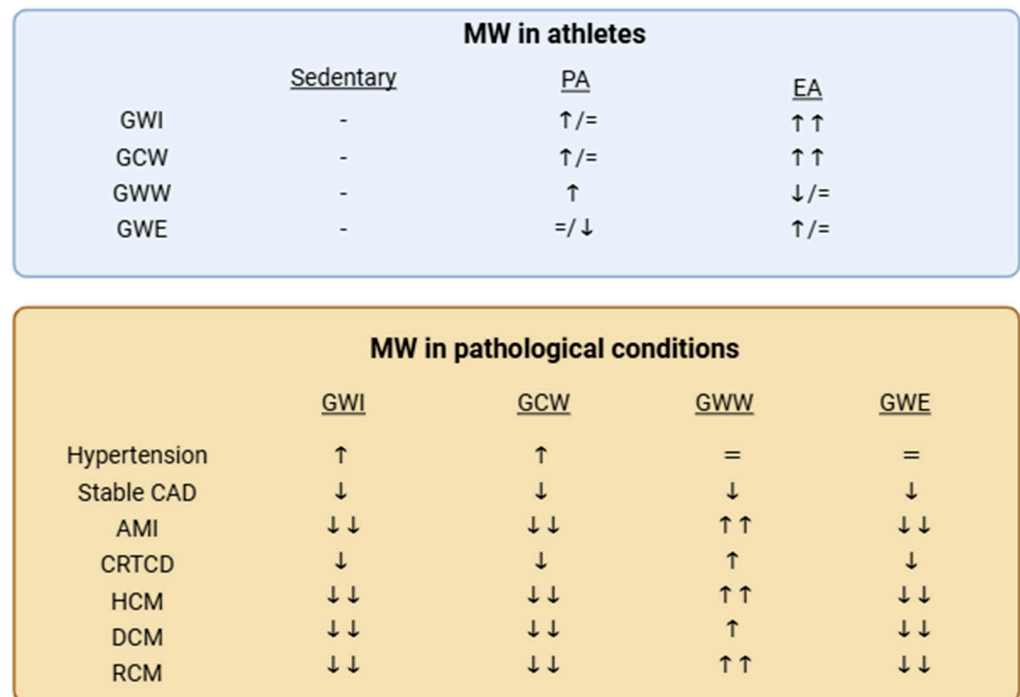


Figure 1. Relative differences in myocardial work indices (GWI, GCW, GWW, GWE) across sedentary individuals, endurance athletes, power athletes, and pathological conditions. Arrows indicate the direction of change compared with sedentary controls (↑ increase, ↓ decrease, = no change).

6. Future Directions and Limitations

The expanding use of MW in both clinical and athletic populations is closely linked to technological advances in echocardiography and computational modeling. Automated contouring and improved strain-tracking algorithms applied to the newest echo machines are reducing inter-operator variability and increasing reproducibility, enabling increasingly precise and reliable measures [75]. Emerging three-dimensional (3D) echocardiography and artificial intelligence (AI)-assisted image analysis may further improve the temporal

and spatial resolution of MW indices, making them more reliable for dynamic cardiac assessment, even during exercise or stress echocardiography.

Portable and wearable imaging devices, although still under development, represent a particularly exciting frontier. These technologies could enable real-time MW analysis during training or competition, providing unprecedented insights into cardiac function in field conditions. Such advancements would support continuous monitoring and early detection of abnormal myocardial responses to extreme training loads [76].

The integration of AI in echocardiography has the potential to transform the field by enhancing diagnostic accuracy, efficiency, and accessibility [77–79]. Myocardial work analysis requires complex echocardiographic imaging and manual calculations, which can be time-consuming and operator-dependent. AI, particularly through machine learning and deep learning algorithms, can automate image acquisition, segmentation, and interpretation, enabling more accurate and reproducible measurements. Moreover, AI can integrate diverse patient data, including imaging, hemodynamic parameters, and clinical history, to generate personalized insights into myocardial work patterns, aiding in risk stratification and treatment planning. Nevertheless, robust clinical validation and regulatory oversight remain essential before widespread adoption.

As our understanding of MW improves, its potential integration into comprehensive athletic assessments is becoming more feasible. Unlike static measures such as LV mass or ejection fraction, MW offers a dynamic, load-adjusted index of cardiac performance that reflects both myocardial contractility and energy consumption. Its application could thus provide tailored insights into how an athlete's heart adapts to specific training regimens.

Studies such as those by Refoyo et al. and Di Gioia et al. demonstrate MW's potential as a standardized tool for evaluating adaptation in diverse athletic profiles, from EA to team sport professionals [21,60].

As previously exposed, differentiating pathological modifications from physiological adaptations in athletes is fundamental and is one of the biggest challenges in sports medicine. A multi-modality approach involving ECG, echocardiogram, cardiac MRI, and other imaging methods is a valid approach to stratify cardiovascular risk more accurately [80]. The integration of MW in this multi-modality approach could help in better identifying structural cardiac diseases and characterizing subclinical myocardial dysfunction, overcoming the limitations of the other techniques. Also in this context, AI could play a crucial role in the integration of data derived from an athlete's history and imaging findings, potentially uncovering new correlations between MW indices and patient outcomes, leading to improved prognostic models and allowing a more precise and personalized approach. Given the growing concern about sudden cardiac death in competitive sports, incorporating MW into pre-participation screening protocols, especially in athletes with borderline findings, may help differentiate physiologic from pathologic hypertrophy or identify subclinical cardiomyopathies [17].

CPET provides detailed information on the global cardiopulmonary response to exercise. The relation between CPET and MW has already been exposed in this work. Combining these two modalities allows for a more nuanced understanding of the interplay between central cardiac mechanics and systemic exercise capacity. As technology advances, the feasibility of performing both assessments in a streamlined clinical workflow is increasing, enhancing their value in routine cardiovascular care. In this way, specifically in the athletes' population, clinicians and sports cardiologists can gain a deeper understanding of how well the heart performs under physical stress and how efficiently it converts energy into effective work. For example, regular exercise training is associated with decreased GLS but increased GWI at rest, and GWI is positively correlated with VO_2/kg [55]. This

approach could help optimize training regimens and detect maladaptive responses to intense physical activity.

In elite athletes, longitudinal tracking of MW may reveal subtle signs of overtraining, deconditioning, or maladaptive remodeling well before overt symptoms or changes in conventional echocardiographic parameters arise. MW may also be useful in training periodization, guiding the balance between endurance and resistance to optimize cardiac workload efficiency while minimizing strain and fatigue. Moreover, integration of MW with other physiological markers (e.g., lactate threshold, heart rate variability) may provide a more comprehensive understanding of an athlete's cardiovascular fitness and adaptation.

There is also growing interest in using MW to personalize return-to-play strategies following illness (e.g., myocarditis, long COVID), cardiac injury, or surgical interventions. MW could help define safer thresholds for re-engagement with competitive activity by tracking recovery of cardiac energetics. Figure 2 provides a graphical summary of MW applications in athletes, highlighting their role in performance monitoring, disease screening, and comprehensive cardiovascular assessment.

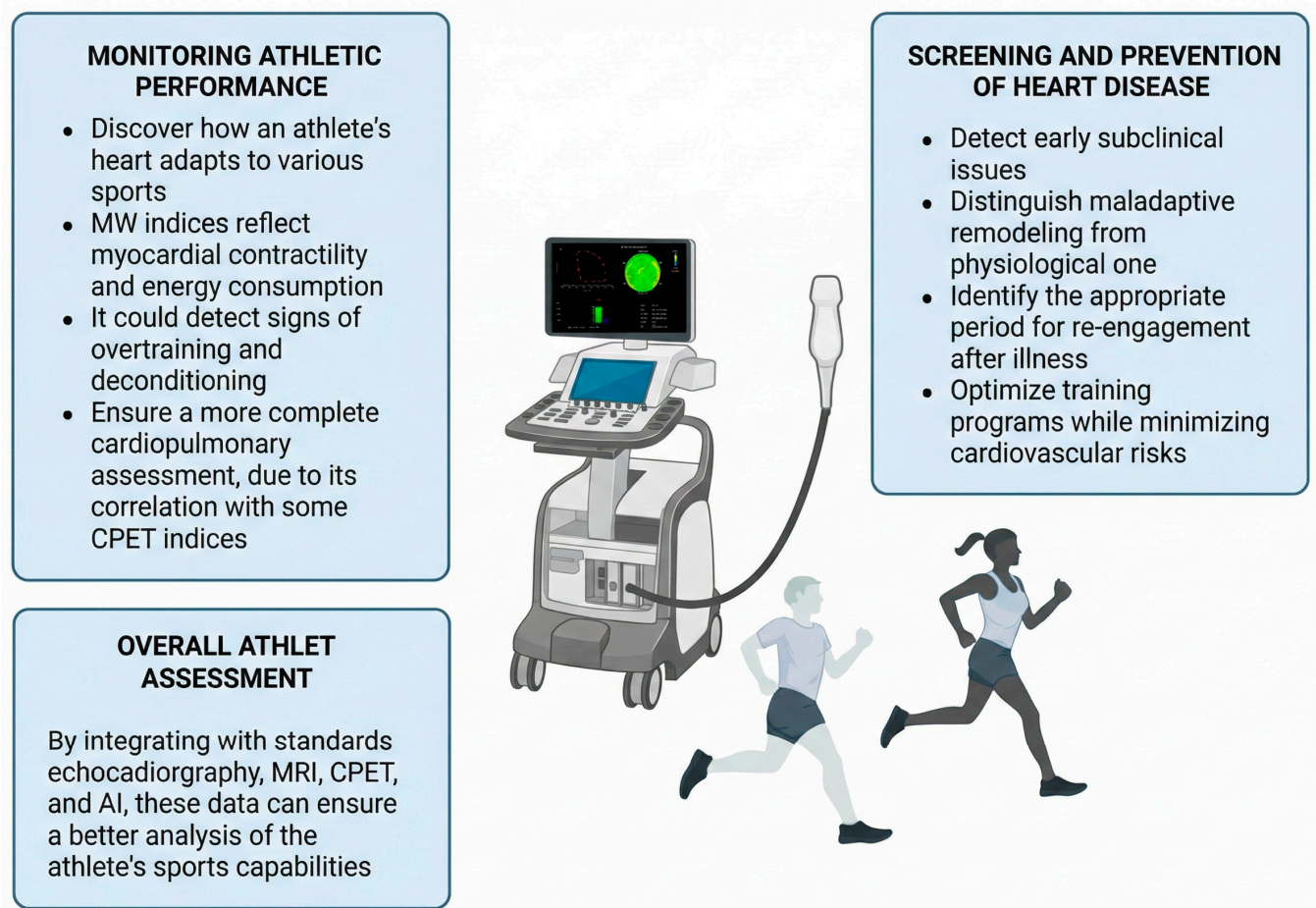


Figure 2. Potential applications of Myocardial Work (MW) parameters in understanding and monitoring cardiac adaptation and performance in athletes.

Despite its promise, the clinical adoption of MW in sports cardiology is not without limitations.

At present, MW assessment is performed with a vendor-specific algorithm, restricting accessibility only to structures provided by these facilities and reducing standardization.

Another limitation is the paucity of large normative datasets for MW in diverse athletic populations. Most current data are derived from small, homogenous cohorts. We lack robust sex-, age-, and ethnicity-specific MW reference ranges in athletes. There is also a

limited understanding of how MW changes across the athletic lifecycle, from youth training to retirement, and in female athletes, who are historically underrepresented in cardiac performance research.

Importantly, because of the limited availability of large comparative datasets, no validated cut-off values currently exist for MW that can reliably distinguish sedentary individuals, strength/power athletes, endurance athletes, and patients with pathological cardiac conditions. Although some studies describe general trends across these groups, the absence of standardized reference ranges and multicenter validation prevents the establishment of diagnostic thresholds. Consequently, MW cannot yet be used to classify athletic phenotypes or differentiate physiological from pathological remodeling based on absolute numerical values.

Moreover, long-term, prospective studies are needed to validate whether MW can truly predict performance outcomes, prevent injury, or identify at-risk individuals with higher sensitivity and specificity than traditional imaging parameters. More prospective, longitudinal studies are needed to validate the prognostic utility of MW in distinguishing physiological from pathological adaptations, especially in relation to arrhythmogenic risks or subclinical myocardial dysfunction. Until then, MW should be interpreted as part of a broader diagnostic and performance-oriented framework.

Additionally, several technical limitations influence the reliability and precision of MW determination, which is based on two moments: the echocardiographic technique and the arterial pressure measurement. Hence, limitations could derive from both.

6.1. Technical Limitations Related to the Echocardiographic Technique

MW relies on speckle-tracking echocardiography, which inherits several source limitations. Poor acoustic windows, foreshortened apical views, or low frame rates (typically <40 fps) hamper accurate endocardial border delineation and speckle-tracking, leading to unreliable longitudinal strain and thus erroneous MW indices [26]. Current methods use exclusively longitudinal deformation from 2D images, ignoring radial and circumferential myocardial work, which may lead to underestimation [81]. The algorithm presumes uniform wall thickness, segmental geometry, and pressure distribution. Variations, like regional hypertrophy, curved chambers, or conduction delays (e.g., LBBB), can skew results. Finally, significant rhythm irregularities (e.g., atrial fibrillation, tachycardia) and anatomical anomalies (e.g., chest wall deformities) impair strain trace acquisition, prompting exclusion from most MW studies [26].

6.2. Limitations Related to Arterial Pressure Measurement

MW uses non-invasive brachial cuff pressure as a surrogate for LV systolic pressure, an assumption that may be inaccurate in cases with significant aortic or LV outflow tract gradients (e.g., severe aortic stenosis, LV outflow tract obstruction), potentially leading to under- or overestimation. Pressure measurements must coincide with echocardiographic acquisition (e.g., patient position, resting state) to accurately reflect afterload during imaging; any mismatch can distort MW calculations. Additionally, this method considers only systolic function and does not account for diastolic (end-diastolic) pressure, which may misrepresent conditions associated with altered preload, such as volume overload [82]. Although MW aims to account for afterload, it remains indirectly dependent on non-invasive blood pressure measurements, which may not accurately reflect central aortic pressures or dynamic changes during exercise. This limitation is particularly relevant in athletes, whose peripheral vascular compliance can vary with body position, hydration, and training state. The method also assumes that LV pressure equals aortic pressure, ignoring transvalvular gradients. While adjusted approaches incorporating mean gradients to es-

timate non-invasive LV systolic pressure have been proposed, they are not yet routinely applied [83].

Because of the intrinsic technical variability of MW measurements and the absence of population-specific reference values, the incorporation of MW into routine echocardiographic reporting remains limited, and its interpretation should be integrated cautiously within a broader multimodal assessment.

Table 3 outlines key limitations in the application of MW and proposes strategies to mitigate these issues in both clinical and athletic settings.

Table 3. Technical limitations of MW analysis and potential strategies to overcome them.

Limitations	Description of Limitation	Strategies
Quality of echocardiographic images	<ul style="list-style-type: none"> • Suboptimal acoustic windows • Foreshortened apical views • Frame rates below 40 fps • Echocardiographic artifacts 	<ul style="list-style-type: none"> • Using high-frequency probes • Optimizing acquisition settings, with recommended frame rates of at least 60–80 fps • Structured operator training programs
Blood pressure measurement	<ul style="list-style-type: none"> • Variability due to body position, hydration, or exercise state. • Brachial pressure may not reflect central aortic pressure • Does not account for transvalvular gradients 	<ul style="list-style-type: none"> • Synchronization of blood pressure measurements and echocardiographic acquisition under the same conditions • Integration of non-invasive central pressure assessment • Application of Doppler-derived gradient corrections in valvular heart disease
Dependence on 2D models	<ul style="list-style-type: none"> • Only longitudinal deformation is considered, ignoring radial and circumferential components 	<ul style="list-style-type: none"> • Development and application of 3D algorithms for MW • Integration with CMR (Cardiac MRI) in complex cases for validation
Vendor-specific algorithm	<ul style="list-style-type: none"> • Mw analysis is only accessible to the structures provided by the single algorithm • Lack of standardization 	<ul style="list-style-type: none"> • Promotion of consensus guidelines to standardize MW analysis • Multicenter validation studies
Clinical applicability	<ul style="list-style-type: none"> • Lack of large normative datasets (by sex, age, ethnicity, athletic level) 	<ul style="list-style-type: none"> • Creation of international registries and multicenter databases • Longitudinal studies in diverse athlete and non-athlete populations • Development of dynamic reference ranges (adjusted for age and training load)

7. Conclusions

MW represents a significant advance in cardiac imaging, offering a comprehensive and load-adjusted assessment of myocardial function. Unlike GLS, MW integrates myocardial deformation with estimated intraventricular pressure, providing a more physiologically meaningful index of performance and energy efficiency.

In athletes, MW is particularly valuable for distinguishing adaptive remodeling from pathology. Quantifying myocardial energy expenditure helps differentiate the physiological

“athlete’s heart” from cardiomyopathic changes and correlates with aerobic capacity and training intensity. Emerging evidence also suggests that MW alterations may precede overt dysfunction, highlighting their promise for early detection and risk stratification.

As the understanding of myocardial energetics grows, MW could become a key tool for integrative athlete monitoring, guiding individualized training, preventing maladaptive adaptations, and supporting long-term cardiovascular health. Future efforts should focus on standardizing acquisition protocols, establishing normative datasets across populations, and validating MW in large-scale athletic cohorts. Meeting these goals will position MW as a central component in the science and practice of sports cardiology.

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References

1. Pelliccia, A.; Sharma, S.; Gati, S.; Bäck, M.; Börjesson, M.; Caselli, S.; Collet, J.-P.; Corrado, D.; Drezner, J.A.; Halle, M.; et al. 2020 ESC Guidelines on Sports Cardiology and Exercise in Patients with Cardiovascular Disease. *Eur. Heart J.* **2021**, *42*, 17–96. [[CrossRef](#)]
2. Segreti, A.; Celeski, M.; Guerra, E.; Crispino, S.P.; Vespasiano, F.; Buzzelli, L.; Fossati, C.; Papalia, R.; Pigozzi, F.; Grigioni, F. Effects of Environmental Conditions on Athlete’s Cardiovascular System. *J. Clin. Med.* **2024**, *13*, 4961. [[CrossRef](#)]
3. Sharma, S.; Merghani, A.; Mont, L. Exercise and the Heart: The Good, the Bad, and the Ugly. *Eur. Heart J.* **2015**, *36*, 1445–1453. [[CrossRef](#)]
4. Pluim, B.M.; Zwinderman, A.H.; Van Der Laarse, A.; Van Der Wall, E.E. The Athlete’s Heart: A Meta-Analysis of Cardiac Structure and Function. *Circulation* **2000**, *101*, 336–344. [[CrossRef](#)]
5. La Gerche, A.; Wasfy, M.M.; Brosnan, M.J.; Claessen, G.; Fatkin, D.; Heidbuchel, H.; Baggish, A.L.; Kovacic, J.C. The Athlete’s Heart—Challenges and Controversies. *J. Am. Coll. Cardiol.* **2022**, *80*, 1346–1362. [[CrossRef](#)]
6. Martinez, M.W.; Kim, J.H.; Shah, A.B.; Phelan, D.; Emery, M.S.; Wasfy, M.M.; Fernandez, A.B.; Bunch, T.J.; Dean, P.; Danielian, A.; et al. Exercise-Induced Cardiovascular Adaptations and Approach to Exercise and Cardiovascular Disease. *J. Am. Coll. Cardiol.* **2021**, *78*, 1453–1470. [[CrossRef](#)]
7. Di Gioia, G.; Ferrera, A.; Mango, F.; Maestrini, V.; Monosilio, S.; Pelliccia, A.; Squeo, M.R. The Spectrum of Eccentric Left Ventricular Hypertrophy in Endurance Sports Disciplines. *Int. J. Cardiovasc. Imaging* **2025**, *41*, 1407–1422. [[CrossRef](#)]
8. D’Andrea, A.; Carbone, A.; Radmilovic, J.; Russo, V.; Fabiani, D.; Maio, M.D.; Ilardi, F.; Giallauria, F.; Caputo, A.; Cirillo, T.; et al. Myocardial Work Efficiency in Physiologic Left Ventricular Hypertrophy of Power Athletes. *J. Cardiovasc. Echogr.* **2022**, *32*, 154–159. [[CrossRef](#)]
9. Pelliccia, A.; Caselli, S.; Sharma, S.; Basso, C.; Bax, J.J.; Corrado, D.; D’Andrea, A.; D’Ascenzi, F.; Di Paolo, F.M.; Edvardsen, T.; et al. European Association of Preventive Cardiology (EAPC) and European Association of Cardiovascular Imaging (EACVI) Joint Position Statement: Recommendations for the Indication and Interpretation of Cardiovascular Imaging in the Evaluation of the Athlete’s Heart. *Eur. Heart J.* **2018**, *39*, 1949–1969. [[CrossRef](#)]
10. Fanale, V.; Segreti, A.; Fossati, C.; Di Gioia, G.; Coletti, F.; Crispino, S.P.; Picarelli, F.; Antonelli Incalzi, R.; Papalia, R.; Pigozzi, F.; et al. Athlete’s ECG Made Easy: A Practical Guide to Surviving Everyday Clinical Practice. *J. Cardiovasc. Dev. Dis.* **2024**, *11*, 303. [[CrossRef](#)]
11. Sharma, S.; Drezner, J.A.; Baggish, A.; Papadakis, M.; Wilson, M.G.; Prutkin, J.M.; La Gerche, A.; Ackerman, M.J.; Borjesson, M.; Salerno, J.C.; et al. International Recommendations for Electrocardiographic Interpretation in Athletes. *Eur. Heart J.* **2018**, *39*, 1466–1480. [[CrossRef](#)]

12. Celeski, M.; Segreti, A.; Crisci, F.; Cricco, R.; Piscione, M.; Di Gioia, G.; Nusca, A.; Fossati, C.; Pigozzi, F.; Ussia, G.P.; et al. The Role of Cardiac Troponin and Other Emerging Biomarkers Among Athletes and Beyond: Underlying Mechanisms, Differential Diagnosis, and Guide for Interpretation. *Biomolecules* **2024**, *14*, 1630. [CrossRef]
13. Celeski, M.; Segreti, A.; Piscione, M.; Monticelli, L.M.; Di Gioia, G.; Fossati, C.; Ussia, G.P.; Pigozzi, F.; Grigioni, F. The Current Paradigm of Cardiac Troponin Increase Among Athletes. *Monaldi Arch. Chest Dis.* **2025**, *95*, 2878. [CrossRef]
14. Boe, E.; Skulstad, H.; Smiseth, O.A. Myocardial Work by Echocardiography: A Novel Method Ready for Clinical Testing. *Eur. Heart J. Cardiovasc. Imaging* **2019**, *20*, 18–20. [CrossRef]
15. Suga, H.; Hayashi, T.; Suehiro, S.; Hisano, R.; Shirahata, M.; Ninomiya, I. Equal Oxygen Consumption Rates of Isovolumic and Ejecting Contractions with Equal Systolic Pressure-Volume Areas in Canine Left Ventricle. *Circ. Res.* **1981**, *49*, 1082–1091. [CrossRef]
16. Russell, K.; Eriksen, M.; Aaberge, L.; Wilhelmsen, N.; Skulstad, H.; Gjesdal, O.; Edvardsen, T.; Smiseth, O.A. Assessment of Wasted Myocardial Work: A Novel Method to Quantify Energy Loss Due to Uncoordinated Left Ventricular Contractions. *Am. J. Physiol. Heart Circ. Physiol.* **2013**, *305*, H996–H1003. [CrossRef]
17. Marzlin, N.; Hays, A.G.; Peters, M.; Kaminski, A.; Roemer, S.; O’Leary, P.; Kroboth, S.; Harland, D.R.; Khandheria, B.K.; Tajik, A.J.; et al. Myocardial Work in Echocardiography. *Circ. Cardiovasc. Imaging* **2023**, *16*, 198–214. [CrossRef]
18. Oxborough, D.; Augustine, D.; Gati, S.; George, K.; Harkness, A.; Mathew, T.; Papadakis, M.; Ring, L.; Robinson, S.; Sandoval, J.; et al. A Guideline Update for the Practice of Echocardiography in the Cardiac Screening of Sports Participants: A Joint Policy Statement from the British Society of Echocardiography and Cardiac Risk in the Young. *Echo Res. Pract.* **2018**, *5*, G1–G10. [CrossRef]
19. Palermi, S.; Serio, A.; Vecchiato, M.; Sirico, F.; Gambardella, F.; Ricci, F.; Iodice, F.; Radmilovic, J.; Russo, V.; D’Andrea, A. Potential Role of an Athlete-Focused Echocardiogram in Sports Eligibility. *World J. Cardiol.* **2021**, *13*, 271–297. [CrossRef]
20. Segreti, A.; Celeski, M.; Monticelli, L.M.; Perillo, A.; Crispino, S.P.; Di Gioia, G.; Cammalleri, V.; Fossati, C.; Mega, S.; Papalia, R.; et al. Mitral and Tricuspid Valve Disease in Athletes. *J. Clin. Med.* **2023**, *12*, 3562. [CrossRef]
21. Refoyo, E.; Troya, J.; De La Fuente, A.; Beltrán, A.; Celada, O.L.; Díaz-González, L.; Pedrero-Tomé, R.; García-Yébenes, M.; Villalón, J.M. Myocardial Work Index in Professional Football Players: A Novel Method for Assessment of Cardiac Adaptation. *J. Clin. Med.* **2023**, *12*, 3059. [CrossRef]
22. Marwick, T.H. Ejection Fraction Pros and Cons. *J. Am. Coll. Cardiol.* **2018**, *72*, 2360–2379. [CrossRef]
23. Konstam, M.A.; Abboud, F.M. Ejection Fraction: Misunderstood and Overrated (Changing the Paradigm in Categorizing Heart Failure). *Circulation* **2017**, *135*, 717–719. [CrossRef]
24. Amzulescu, M.S.; De Craene, M.; Langet, H.; Pasquet, A.; Vancraeynest, D.; Pouleur, A.C.; Vanoverschelde, J.L.; Gerber, B.L. Myocardial Strain Imaging: Review of General Principles, Validation, and Sources of Discrepancies. *Eur. Heart J. Cardiovasc. Imaging* **2019**, *20*, 605–619. [CrossRef]
25. Bastos, M.B.; Burkhoff, D.; Maly, J.; Daemen, J.; Den Uil, C.A.; Ameloot, K.; Lenzen, M.; Mahfoud, F.; Zijlstra, F.; Schreuder, J.J.; et al. Invasive Left Ventricle Pressure–Volume Analysis: Overview and Practical Clinical Implications. *Eur. Heart J.* **2020**, *41*, 1286–1297. [CrossRef]
26. Ilardi, F.; D’Andrea, A.; D’Ascenzi, F.; Bandera, F.; Benfari, G.; Esposito, R.; Malagoli, A.; Mandoli, G.E.; Santoro, C.; Russo, V.; et al. Myocardial Work by Echocardiography: Principles and Applications in Clinical Practice. *J. Clin. Med.* **2021**, *10*, 4521. [CrossRef]
27. Suga, H. Total Mechanical Energy of a Ventricle Model and Cardiac Oxygen Consumption. *Am. J. Physiol. Heart Circ. Physiol.* **1979**, *236*, H498–H505. [CrossRef] [PubMed]
28. Russell, K.; Eriksen, M.; Aaberge, L.; Wilhelmsen, N.; Skulstad, H.; Remme, E.W.; Haugaa, K.H.; Opdahl, A.; Fjeld, J.G.; Gjesdal, O.; et al. A Novel Clinical Method for Quantification of Regional Left Ventricular Pressure–Strain Loop Area: A Non-Invasive Index of Myocardial Work. *Eur. Heart J.* **2012**, *33*, 724–733. [CrossRef]
29. Samset, E.; Healthcare, G. Evaluation of Segmental Myocardial Work in the Left Ventricle. Available online: https://www.gehealthcare.com/-/media/8cab29682ace4ed7841505f813001e33.pdf@line-2@?srsltid=AfmBOop7qrhKkrMsDpOIJvk-1r3YPNOWusaW334bL3mB6ke3wPE_yMiB (accessed on 1 July 2025).
30. Manganaro, R.; Marchetta, S.; Dulgheru, R.; Ilardi, F.; Sugimoto, T.; Robinet, S.; Cimino, S.; Go, Y.Y.; Bernard, A.; Kacharava, G.; et al. Echocardiographic Reference Ranges for Normal Non-Invasive Myocardial Work Indices: Results from the EACVI NORRE Study. *Eur. Heart J. Cardiovasc. Imaging* **2019**, *20*, 582–590. [CrossRef]
31. Chan, J.; Edwards, N.F.A.; Khandheria, B.K.; Shiino, K.; Sabapathy, S.; Anderson, B.; Chamberlain, R.; Scalia, G.M. A New Approach to Assess Myocardial Work by Non-Invasive Left Ventricular Pressure–Strain Relations in Hypertension and Dilated Cardiomyopathy. *Eur. Heart J. Cardiovasc. Imaging* **2019**, *20*, 31–39. [CrossRef]
32. Tadic, M.; Cuspidi, C.; Pencic, B.; Grassi, G.; Celic, V. Myocardial Work in Hypertensive Patients with and without Diabetes: An Echocardiographic Study. *J. Clin. Hypertens.* **2020**, *22*, 2121–2127. [CrossRef]

33. Zhang, J.; Liu, Y.; Deng, Y.; Zhu, Y.; Sun, R.; Lu, S. Non-Invasive Global and Regional Myocardial Work Predicts High-Risk Stable Coronary Artery Disease Patients with Normal Segmental Wall Motion and Left Ventricular Function. *Front. Cardiovasc. Med.* **2021**, *8*, 711547. [[CrossRef](#)]
34. Boe, E.; Russell, K.; Eek, C.; Eriksen, M.; Remme, E.W.; Smiseth, O.A.; Skulstad, H. Non-Invasive Myocardial Work Index Identifies Acute Coronary Occlusion in Patients with Non-ST-Segment Elevation-Acute Coronary Syndrome. *Eur. Heart J. Cardiovasc. Imaging* **2015**, *16*, 1247–1255. [[CrossRef](#)]
35. Jin, W.; Wang, L.; Zhu, T.; Ma, Y.; Yu, C.; Zhang, F. Usefulness of Echocardiographic Myocardial Work in Evaluating the Microvascular Perfusion in STEMI Patients After Revascularization. *BMC Cardiovasc. Disord.* **2022**, *22*, 218. [[CrossRef](#)] [[PubMed](#)]
36. Lyon, A.R.; López-Fernández, T.; Couch, L.S.; Asteggiano, R.; Aznar, M.C.; Bergler-Klein, J.; Boriani, G.; Cardinale, D.; Cordoba, R.; Cosyns, B.; et al. 2022 ESC Guidelines on Cardio-Oncology Developed in Collaboration with the European Hematology Association (EHA), the European Society for Therapeutic Radiology and Oncology (ESTRO) and the International Cardio-Oncology Society (IC-OS). *Eur. Heart J.* **2022**, *43*, 4229–4361. [[CrossRef](#)]
37. Zhan, J.; Van Den Eynde, J.; Cordrey, K.; Long, R.; Danford, D.A.; Hays, A.G.; Barnes, B.T.; Kutty, S. Deterioration in Myocardial Work Indices Precedes Changes in Global Longitudinal Strain Following Anthracycline Chemotherapy. *Int. J. Cardiol.* **2022**, *363*, 171–178. [[CrossRef](#)] [[PubMed](#)]
38. Guan, J.; Bao, W.; Xu, Y.; Yang, W.; Li, M.; Xu, M.; Zhang, Y.; Zhang, M. Assessment of Myocardial Work in Cancer Therapy-Related Cardiac Dysfunction and Analysis of CTRCD Prediction by Echocardiography. *Front. Pharmacol.* **2021**, *12*, 770580. [[CrossRef](#)]
39. Calvillo-Argüelles, O.; Thampinathan, B.; Somerset, E.; Shalmon, T.; Amir, E.; Steve Fan, C.-P.; Moon, S.; Abdel-Qadir, H.; Thevakumaran, Y.; Day, J.; et al. Diagnostic and Prognostic Value of Myocardial Work Indices for Identification of Cancer Therapy-Related Cardiotoxicity. *JACC Cardiovasc. Imaging* **2022**, *15*, 1361–1376. [[CrossRef](#)]
40. Galli, E.; Vitel, E.; Schnell, F.; Le Rolle, V.; Hubert, A.; Lederlin, M.; Donal, E. Myocardial Constructive Work Is Impaired in Hypertrophic Cardiomyopathy and Predicts Left Ventricular Fibrosis. *Echocardiography* **2019**, *36*, 74–82. [[CrossRef](#)]
41. Xiao, C.; Zhao, X.; Sun, L.; Zhang, F. Evaluation of Global and Regional Myocardial Work in Hypertrophic Cardiomyopathy Patients by Left Ventricular Pressure-Strain Loop. *BMC Cardiovasc. Disord.* **2023**, *23*, 479. [[CrossRef](#)]
42. Hiemstra, Y.L.; van der Bijl, P.; El Mahdiui, M.; Bax, J.J.; Delgado, V.; Marsan, N.A. Myocardial Work in Nonobstructive Hypertrophic Cardiomyopathy: Implications for Outcome. *J. Am. Soc. Echocardiogr.* **2020**, *33*, 1201–1208. [[CrossRef](#)]
43. Jacquemyn, X.; Dryer, R.; Cordrey, K.; Long, R.; Danford, D.A.; Kutty, S.; Barnes, B.T. Myocardial Work in Children with Hypertrophic Cardiomyopathy: Longitudinal Evaluation and Prognostic Implications. *JACC Adv.* **2025**, *4*, 101885. [[CrossRef](#)]
44. Cui, C.; Li, Y.; Liu, Y.; Huang, D.; Hu, Y.; Wang, Y.; Ma, L.; Liu, L. Association Between Echocardiographic Non-Invasive Myocardial Work Indices and Myocardial Fibrosis in Patients with Dilated Cardiomyopathy. *Front. Cardiovasc. Med.* **2021**, *8*, 704251. [[CrossRef](#)]
45. Hedwig, F.; Nemchyna, O.; Stein, J.; Knosalla, C.; Merke, N.; Knebel, F.; Hagendorff, A.; Schoenrath, F.; Falk, V.; Knierim, J. Myocardial Work Assessment for the Prediction of Prognosis in Advanced Heart Failure. *Front. Cardiovasc. Med.* **2021**, *8*, 691611. [[CrossRef](#)] [[PubMed](#)]
46. Ródenas-Alesina, E.; Lozano-Torres, J.; Badia-Molins, C.; Tobías-Castillo, P.E.; Rodríguez-Palomares, J.F.; Ferreira-González, I. Association Between Global Myocardial Work Index and Outcomes in Nonischemic Dilated Cardiomyopathy. *J. Cardiovasc. Med.* **2025**, *26*, 547–551. [[CrossRef](#)]
47. Vrettos, A.; Monteiro, R.P.; Triantafyllou, M.; Gul, U.; Bhattacharyya, S.; Lopes, L.R.; Antonopoulos, A.; Protonotarios, A.; Lloyd, G.; Gossios, T.; et al. Early Detection of Subclinical Myocardial Dysfunction in Familial Dilated Cardiomyopathy Using Myocardial Work Analysis. *Diagnostics* **2025**, *15*, 2363. [[CrossRef](#)]
48. Li, X.; Liu, X.; Feng, X.; Guo, T.; Liu, G.; Wu, D.; Liu, Y.; Lai, J.; Liu, Y.; Lin, X.; et al. Prognostic Implications of Multiple Chamber Longitudinal Strains and Myocardial Work in Restrictive Cardiomyopathy. *Sci. Rep.* **2025**, *15*, 12504. [[CrossRef](#)]
49. Guazzi, M.; Adams, V.; Conraads, V.; Halle, M.; Mezzani, A.; Vanhees, L.; Arena, R.; Fletcher, G.F.; Forman, D.E.; Kitzman, D.W.; et al. Clinical Recommendations for Cardiopulmonary Exercise Testing Data Assessment in Specific Patient Populations. *Circulation* **2012**, *126*, 2261–2274. [[CrossRef](#)]
50. Petek, B.J.; Gustus, S.K.; Wasfy, M.M. Cardiopulmonary Exercise Testing in Athletes: Expect the Unexpected. *Curr. Treat. Options Cardio. Med.* **2021**, *23*, 49. [[CrossRef](#)]
51. Segreti, A.; Picarelli, F.; Di Gioia, G.; Coletti, F.; Crispino, S.P.; Fanale, V.; Fossati, C.; Antonelli Incalzi, R.; Pigozzi, F.; Grigioni, F. Athlete’s Heart or Heart Disease in the Athlete? Evaluation by Cardiopulmonary Exercise Testing. *J. Sports Med. Phys. Fit.* **2023**, *63*, 873–890. [[CrossRef](#)] [[PubMed](#)]
52. Pelliccia, A.; Heidbüchel, H.; Corrado, D.; Sharma, S.; Börjesson, M. Criteria and Considerations Relative to Safe Participation in Sport for Athletes with Cardiac Abnormalities. In *The ESC Textbook of Sports Cardiology*; Pelliccia, A., Heidbüchel, H., Corrado, D., Börjesson, M., Sharma, S., Pelliccia, A., Heidbüchel, H., Corrado, D., Borjesson, M., Sharma, S., Eds.; Oxford University Press: Oxford, UK, 2019. [[CrossRef](#)]

53. Arena, R.; Ozemek, C. Intracardiac Multimorbidity: Assessing Right Ventricular Function in Left-Sided Heart Failure Through Cardiopulmonary Exercise Testing. *Expert. Rev. Cardiovasc. Ther.* **2019**, *17*, 331–333. [[CrossRef](#)]
54. Zhao, K.; Liu, Y.; Dong, L.; Gao, B. Echocardiographic Myocardial Work in Pre-Adolescent Male Basketball Players: A Comparison with Cardiopulmonary Exercise Test-Derived Aerobic Capacity. *Front. Physiol.* **2022**, *13*, 913623. [[CrossRef](#)] [[PubMed](#)]
55. Tokodi, M.; Oláh, A.; Fábíán, A.; Lakatos, B.K.; Hizoh, I.; Ruppert, M.; Sayour, A.A.; Barta, B.A.; Kiss, O.; Sydó, N.; et al. Novel Insights into the Athlete's Heart: Is Myocardial Work the New Champion of Systolic Function? *Eur. Heart J. Cardiovasc. Imaging* **2022**, *23*, 188–197. [[CrossRef](#)]
56. Segreti, A.; Fossati, C.; Monticelli, L.M.; Valente, D.; Polito, D.; Guerra, E.; Zampoli, A.; Albimonti, G.; Zampogna, B.; Vasta, S.; et al. Changes in Cardiopulmonary Capacity Parameters After Surgery: A Pilot Study Exploring the Link Between Heart Function and Knee Surgery. *J. Funct. Morphol. Kinesiol.* **2024**, *9*, 172. [[CrossRef](#)]
57. Erevik, C.B.; Kleiven, Ø.; Frøysa, V.; Bjørkavoll-Bergseth, M.; Chivulescu, M.; Klæboe, L.G.; Dejgaard, L.; Auestad, B.; Skadberg, Ø.; Melberg, T.; et al. Myocardial Inefficiency Is an Early Indicator of Exercise-Induced Myocardial Fatigue. *Front. Cardiovasc. Med.* **2023**, *9*, 1081664. [[CrossRef](#)]
58. Kandels, J.; Stöbe, S.; Kogel, A.; Hepp, P.; Riepenhof, H.; Droste, J.N.; Stoeggel, T.; Marshall, R.P.; Rudolph, U.; Laufs, U.; et al. Effect of Maximum Exercise on Left Ventricular Deformation and Its Correlation with Cardiopulmonary Exercise Capacity in Competitive Athletes. *Echo Res. Pract.* **2023**, *10*, 17. [[CrossRef](#)] [[PubMed](#)]
59. D'Andrea, A.; Cameli, M.; Carbone, A.; Mandoli, G.E.; Santoro, C.; Evola, V.; Bandera, F.; D'Ascenzi, F.; Bossone, E.; Galderisi, M.; et al. Speckle Tracking Evaluation in Endurance Athletes: The "Optimal" Myocardial Work. *Int. J. Cardiovasc. Imaging* **2020**, *36*, 1679–1688. [[CrossRef](#)]
60. Di Gioia, G.; Ferrera, A.; Mango, F.; Ortolina, D.; Maestrini, V.; Monosilio, S.; Paoletti, G.; Lemme, E.; Squeo, M.R.; Pelliccia, A. Correlation Between Myocardial Work Indices and Main Echocardiographic and Cardiopulmonary Exercise Stress Test Parameters in Olympic Endurance Athletes. *Eur. J. Appl. Physiol.* **2025**, *125*, 2939–2949. [[CrossRef](#)] [[PubMed](#)]
61. Borzi, D.D.; Saladino, S.; Losi, V.; Faro, D.C.; Monte, I.P. Strain and Myocardial Work Index During Echo Exercise to Evaluate Myocardial Function in Athletes. *J. Cardiovasc. Echogr.* **2022**, *32*, 82–88. [[CrossRef](#)]
62. Avula, H.R.; Leong, T.K.; Lee, K.K.; Sung, S.H.; Go, A.S. Long-Term Outcomes of Adults with Heart Failure by Left Ventricular Systolic Function Status. *Am. J. Cardiol.* **2018**, *122*, 1008–1016. [[CrossRef](#)]
63. Smiseth, O.A.; Torp, H.; Opdahl, A.; Haugaa, K.H.; Urheim, S. Myocardial Strain Imaging: How Useful Is It in Clinical Decision Making? *Eur. Heart J.* **2016**, *37*, 1196–1207. [[CrossRef](#)]
64. Gherbesi, E.; Gianstefani, S.; Angeli, F.; Ryabenko, K.; Bergamaschi, L.; Armillotta, M.; Guerra, E.; Tuttolomondo, D.; Gaibazzi, N.; Squeri, A.; et al. Myocardial Strain of the Left Ventricle by Speckle Tracking Echocardiography: From Physics to Clinical Practice. *Echocardiography* **2024**, *41*, e15753. [[CrossRef](#)]
65. Moya, A.; Buytaert, D.; Penicka, M.; Bartunek, J.; Vanderheyden, M. State-of-the-Art: Noninvasive Assessment of Left Ventricular Function Through Myocardial Work. *J. Am. Soc. Echocardiogr.* **2023**, *36*, 1027–1042. [[CrossRef](#)]
66. Papadopoulos, K.; Özden Tok, Ö.; Mitrousi, K.; Ikonomidis, I. Myocardial Work: Methodology and Clinical Applications. *Diagnostics* **2021**, *11*, 573. [[CrossRef](#)]
67. Huang, L.; Ye, L.; Zhang, H.; Zhang, Q.; Ding, G.; Li, C.; Deng, Y.; Yin, L.; Wang, Y. Characteristics of Myocardial Work During Exercise Stress Echocardiography in Healthy Adults. *Front. Cardiovasc. Med.* **2025**, *12*, 1511464. [[CrossRef](#)] [[PubMed](#)]
68. Mont, L.; Elosua, R.; Brugada, J. Endurance Sport Practice as a Risk Factor for Atrial Fibrillation and Atrial Flutter. *Europace* **2008**, *11*, 11–17. [[CrossRef](#)] [[PubMed](#)]
69. Goette, A.; Corradi, D.; Dobrev, D.; Aguinaga, L.; Cabrera, J.-A.; Chugh, S.S.; De Groot, J.R.; Soulat-Dufour, L.; Fenelon, G.; Hatem, S.N.; et al. Atrial Cardiomyopathy Revisited—Evolution of a Concept: A Clinical Consensus Statement of the European Heart Rhythm Association (EHRA) of the ESC, the Heart Rhythm Society (HRS), the Asian Pacific Heart Rhythm Society (APHRS), and the Latin American Heart Rhythm Society (LAHRS). *Europace* **2024**, *26*, euae204. [[CrossRef](#)]
70. Cuspidi, C.; Tadic, M.; Sala, C.; Gherbesi, E.; Grassi, G.; Mancina, G. Left Atrial Function in Elite Athletes: A Meta-analysis of Two-dimensional Speckle Tracking Echocardiographic Studies. *Clin. Cardiol.* **2019**, *42*, 579–587. [[CrossRef](#)]
71. Li, P.; Zhang, Y.; Li, L.; Chen, Y.; Li, Z.; Liu, S.; Hua, S. Assessment of Left Ventricular Systolic Function by Non-Invasive Pressure-Strain Loop Area in Young Male Strength Athletes. *Cardiovasc. Ultrasound* **2020**, *18*, 45. [[CrossRef](#)] [[PubMed](#)]
72. Sengupta, S.; Jain, R.; Burkule, N.; Olet, S.; Khandheria, B.K. Myocardial Work Index: A Novel Method for Assessment of Myocardial Function in South Asian Recreational Athletes. *J. Patient-Centered Res. Rev.* **2020**, *7*, 147–156. [[CrossRef](#)]
73. Da Luz, S.R.; Okawa, R.T.P.; Lopes, W.A. Myocardial Work and Left Ventricular Function: Implications for Aerobic Capacity and Performance in Professional Soccer Athletes. *Sport Sci. Health* **2025**, *21*, 1889–1898. [[CrossRef](#)]
74. Grandperrin, A.; Schnell, F.; Donal, E.; Galli, E.; Hedon, C.; Cazorla, O.; Nottin, S. Specific Alterations of Regional Myocardial Work in Strength-Trained Athletes Using Anabolic Androgenic Steroids Compared to Athletes with Genetic Hypertrophic Cardiomyopathy. *J. Sport Health Sci.* **2023**, *12*, 477–485. [[CrossRef](#)]
75. Gillam, L.D.; Marcoff, L. Echocardiography: Past, Present, and Future. *Circ. Cardiovasc. Imaging* **2024**, *17*, e016517. [[CrossRef](#)]

76. Hu, H.; Huang, H.; Li, M.; Gao, X.; Yin, L.; Qi, R.; Wu, R.S.; Chen, X.; Ma, Y.; Shi, K.; et al. A Wearable Cardiac Ultrasound Imager. *Nature* **2023**, *613*, 667–675. [[CrossRef](#)] [[PubMed](#)]
77. Alsharqi, M.; Woodward, W.J.; Mumith, J.A.; Markham, D.C.; Upton, R.; Leeson, P. Artificial Intelligence and Echocardiography. *Echo Res. Pract.* **2018**, *5*, R115–R125. [[CrossRef](#)] [[PubMed](#)]
78. Hirata, Y.; Kusunose, K. AI in Echocardiography: State-of-the-Art Automated Measurement Techniques and Clinical Applications. *JMA J.* **2025**, *8*, 141–150. [[CrossRef](#)] [[PubMed](#)]
79. Sahashi, Y.; Ouyang, D.; Okura, H.; Kagiya, N. AI-Echocardiography: Current Status and Future Direction. *J. Cardiol.* **2025**, *85*, 458–464. [[CrossRef](#)]
80. Galderisi, M.; Cardim, N.; D'Andrea, A.; Bruder, O.; Cosyns, B.; Davin, L.; Donal, E.; Edvardsen, T.; Freitas, A.; Habib, G.; et al. The Multi-Modality Cardiac Imaging Approach to the Athlete's Heart: An Expert Consensus of the European Association of Cardiovascular Imaging. *Eur. Heart J. Cardiovasc. Imaging* **2015**, *16*, 353–353r. [[CrossRef](#)]
81. Liao, M.; Pan, J.; Liao, T.; Liu, X.; Wang, L. Transthoracic Echocardiographic Assessment of Ventricular Function in Functional Single Ventricle: A Comprehensive Review. *Cardiovasc. Ultrasound* **2025**, *23*, 9. [[CrossRef](#)]
82. Lembo, M.; Santoro, C.; Casciano, O.; Capone, V.; Fedele, T.; Luciano, F.; Canonico, M.; Buonauro, A.; Esposito, R.; Galderisi, M. Impact of Diastolic Blood Pressure on Speckle Tracking Derived Myocardial Work Components in a Population of Normotensive and Untreated Hypertensive Patients. *Eur. Heart J.* **2020**, *41* (Suppl. S2), ehaa946.2700. [[CrossRef](#)]
83. Jain, R.; Bajwa, T.; Roemer, S.; Huisheree, H.; Allaqaband, S.Q.; Kroboth, S.; Perez Moreno, A.C.; Tajik, A.J.; Khandheria, B.K. Myocardial Work Assessment in Severe Aortic Stenosis Undergoing Transcatheter Aortic Valve Replacement. *Eur. Heart J. Cardiovasc. Imaging* **2021**, *22*, 715–721. [[CrossRef](#)] [[PubMed](#)]

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