Assessment of Epicardial Adipose Tissue by Cardiovascular Magnetic Resonance

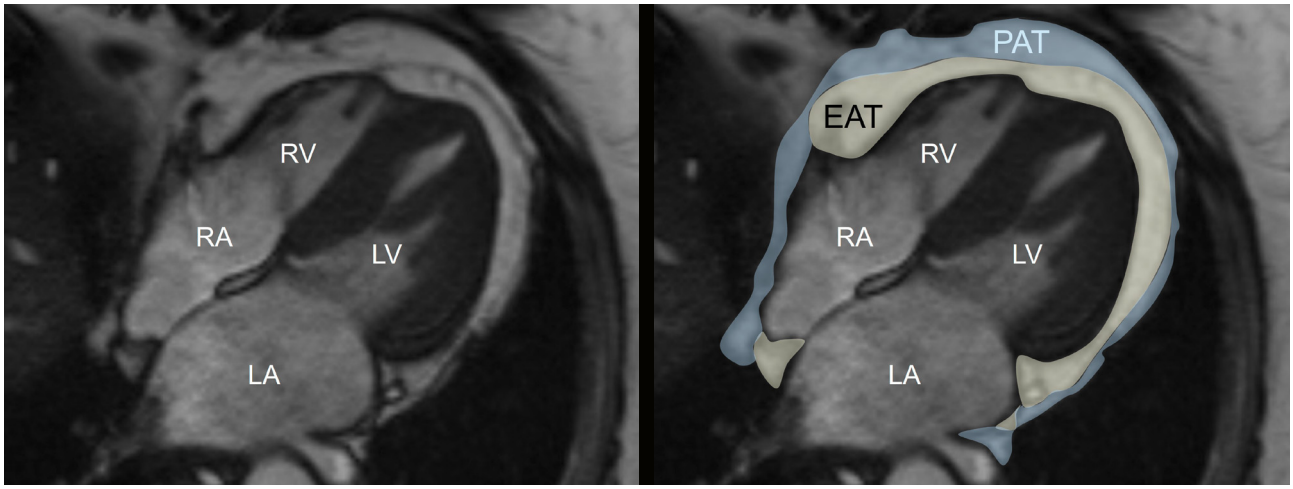
From volumetric quantification to cardiometabolic phenotyping

Judith Gronwald[1,2], Halil Noyan[3,4], Jeanette Schulz-Menger[3,4,5,6], Richard Hickstein[3,4,7], Alexander Schulz[1,2]

[1] Department of Cardiology and Pneumology, University Medical Center Göttingen, Georg-August University, Göttingen, Germany
[2] DZHK (German Centre for Cardiovascular Research), partner site Lower Saxony, Göttingen, Germany
[3] Working Group on Cardiovascular Magnetic Resonance, Experimental and Clinical Research Center, a cooperation between Charité – Universitätsmedizin Berlin and the Max Delbrück Center for Molecular Medicine in the Helmholtz Association, Berlin, Germany
[4] DZHK (German Centre for Cardiovascular Research), partner site Berlin, Germany
[5] Helios Hospital Berlin Buch, Department Cardiology and Nephrology, Berlin, Germany
[6] Deutsches Herzzentrum der Charité Berlin, Germany
[7] Helios Health Institute, Berlin, Germany

1. Epicardial adipose tissue as a cardiometabolic imaging biomarker

Metabolic disorders are major modifiable drivers of cardiovascular disease [1]. However, although body mass index and waist circumference are commonly used as surrogate markers of the metabolic phenotype in clinical practice [2], they primarily capture overall adiposity and provide limited information on abnormal fat distribution and the metabolic activity of adipose tissue. Cardiometabolic phenotyping is therefore increasingly focusing on abnormal fat distribution, particularly the accumulation of visceral fat, and its links to impaired lipid metabolism, inflammation, and organ injury [3]. Epicardial adipose tissue (EAT) is a visceral fat depot of particular interest because it is metabolically active and in close anatomic relationship with the heart [4].



1 Representative 4-chamber view illustrating the different fat tissue compartments surrounding the heart.
EAT = epicardial adipose tissue; LA = left atrium; LV = left ventricle; PAT = pericardial adipose tissue; RA = right atrium; RV = right ventricle

Fat tissue around the heart comprises two distinct compartments separated by the pericardium. EAT lies between the myocardium and the visceral layer of the serous pericardium, while pericardial adipose tissue is located outside the parietal pericardium [4]. Both are often combined as paracardial adipose tissue despite differences in origin, vascular supply, and biological interaction with the heart [5]. For imaging studies, the pericardium forms the decisive boundary between EAT and parietal, pericardial fat (Fig. 1).

Particular interest in EAT arises from its shared microcirculation with the myocardium and its lack of fascial separation from the myocardium or coronary arteries [4]. In healthy individuals, EAT surrounds the coronary arteries, cushioning them, supplying metabolic substrates, contributing to thermal protection, and acting as an endocrine and immune organ [6]. These protective properties are partly attributed to the brown- or beige-fat-like characteristics of EAT, particularly early in life. With aging and exposure to metabolic and inflammatory stressors, including obesity and diabetes, EAT may expand and shift toward a more white adipose-like phenotype [7]. This remodeling can promote inflammation, fibrosis, and lipotoxicity, with potentially adverse effects on the myocardium [4, 8, 9]. Cytokines, adipokines, and free fatty acids released by EAT may act on the adjacent myocardium through paracrine diffusion and on the coronary vessel wall through vasocrine signaling [4]. Fatty infiltration, particularly in the atria [10], and mechanical restraint associated with excessive cardiac fat accumulation [4] provide additional pathways of injury.

EAT is readily visible on routine cardiac imaging, offering imagers an opportunity to extract additional information about cardiometabolic health from images acquired in everyday practice. Cardiovascular magnetic resonance imaging (CMR) enables EAT quantification without radiation. The relationships with ventricular volumes, strain, perfusion, parametric mapping, and late gadolinium enhancement provide additional information. To establish EAT as a clinically useful cardiometabolic marker, its volume, distribution, and phenotype need to be comprehensively characterized. The objective is therefore to integrate these features into cardiometabolic characterization, identify potentially reversible cardiac effects, and determine whether treatment-related changes in EAT volume and composition accompany improved function or lower cardiovascular risk.

2. Clinical relevance across cardiovascular disease

EAT has been associated with the pathophysiology and prognosis of several cardiovascular diseases, although the strength and meaning of these associations vary. Its value is likely greatest when assessed regionally and interpreted with disease-specific imaging markers. Coronary artery disease and atrial fibrillation (AF) provide the most mature evidence, while first data from heart-failure populations and patients with valvular disease illustrate how EAT impacts myocardial structure, perfusion, and function [4, 11]. Figure 2 illustrates three representative CMR cases of patients with different cardiovascular conditions, highlighting the respective EAT burden.

2.1 Coronary artery disease

The coronary arteries traverse EAT, creating a direct interface between adipose tissue and the vessel wall [12]. Increased EAT volume has been associated with coronary calcification, plaque burden, myocardial infarction, incident events, and adverse remodeling after infarction [13, 14]. However, volume is only part of the process: Pericoronary EAT distribution and inflammatory or fibrotic composition may provide valuable information that global volume cannot [15].

Coronary computed tomography (CT) angiography currently offers the most established approach to assessing inflammation-related changes in pericoronary fat through attenuation analysis [16]. Recent advancements in CT technology, notably the clinical introduction of photon-counting CT, have further revolutionized this field. Photon-counting CT provides ultra-high spatial resolution and spectral information, enabling a highly precise evaluation of coronary plaque vulnerability and the composition of surrounding pericoronary adipose tissue [17]. However, emerging CMR approaches offer the potential to extend EAT assessment beyond volumetry [18] and provide a comprehensive, radiation-free evaluation of both adipose tissue and the cardiac tissues that may be affected by it. By combining EAT volume and fat characterization with quantitative myocardial perfusion, ventricular function, and tissue characterization within a single examination, CMR makes it possible to assess EAT in the context of microvascular dysfunction, fibrosis, and myocardial injury, thereby linking the characteristics of the fat depot to its potential cardiac effects.

2.2 Atrial fibrillation and atrial cardiomyopathy

In AF, regional EAT distribution may be more informative than total EAT volume. EAT accumulates around the atrioventricular groove and the pulmonary veins. Periatrial EAT has been associated with incident and persistent AF, atrial remodeling, low-voltage substrate, and recurrence of AF after ablation [11, 19]. Proposed mechanisms include local inflammation, fibrosis, autonomic modulation, conduction heterogeneity, and fatty myocardial infiltration, although cardiometabolic confounding remains important [11].

CMR can combine periatrial fat mapping with atrial volumes and function, ventricular filling, and, where robust, atrial fibrosis or ablation scar [20]. Spatial resolution

remains the principal limitation: The atrial wall and adjacent fat are thin, making focal infiltration vulnerable to partial volume effects [21]. Higher-resolution three-dimensional imaging [22], water-fat separation [23], and motion-robust reconstruction [24] may improve periatrial and intramyocardial fat characterization.

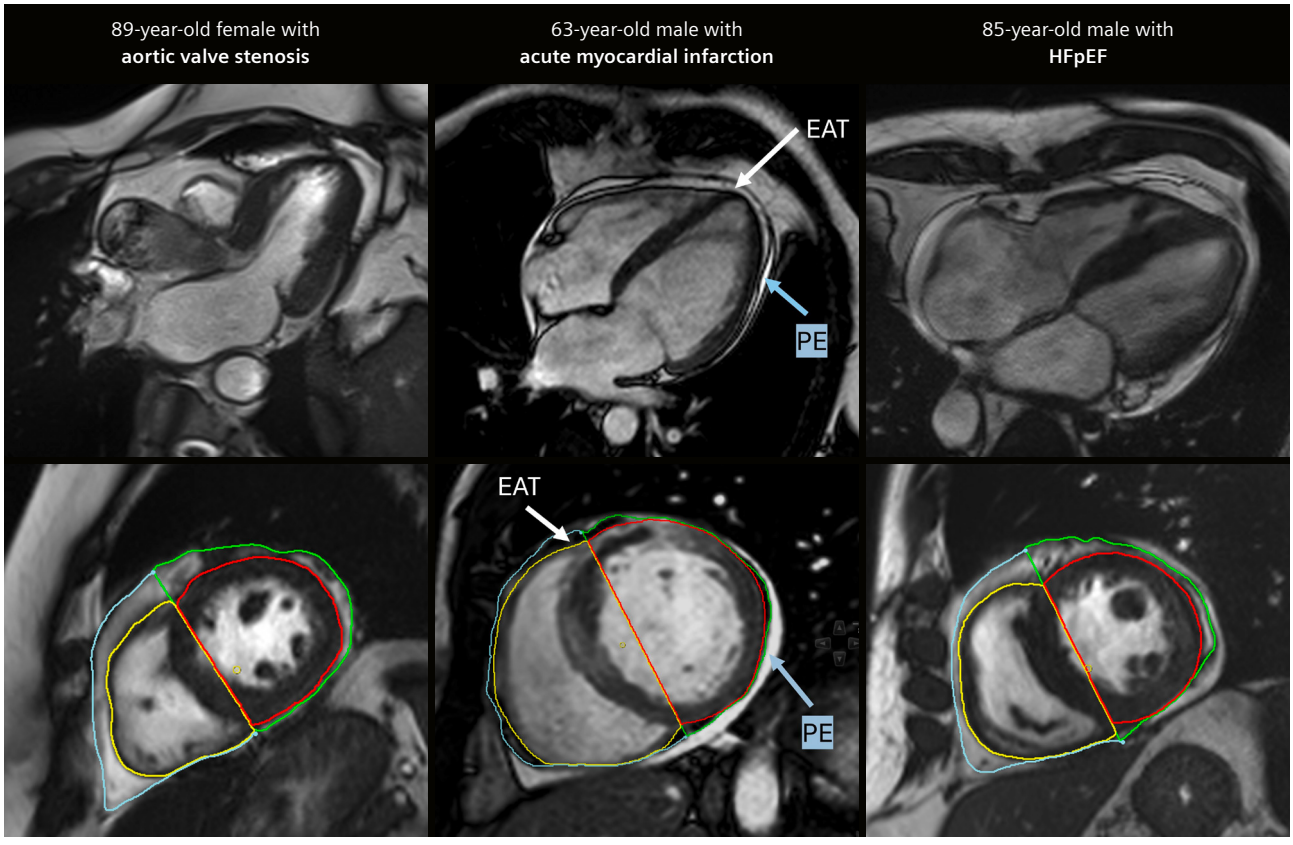
2.3 Heart failure and cardiometabolic myocardial disease

The relationship between EAT and heart failure is phenotype dependent. EAT volume is commonly increased in heart failure with preserved ejection fraction (HFpEF) [25], whereas lower volumes can accompany cachexia and adverse outcomes in advanced heart failure with reduced ejection fraction (HFrEF) [26]. HFrEF may reflect disease severity and loss of physiological cushioning and metabolic support, rather than a benefit from greater adiposity. A single direction-of-risk model is therefore inappropriate across the heart failure spectrum.

In obesity-related HFpEF, greater EAT accumulation is associated with higher cardiac filling pressures and lower exercise capacity [27]. Invasive studies have linked increased EAT to higher right atrial and pulmonary capillary wedge pressures at rest and during exercise, providing a direct clinical connection to congestion and exertional intolerance [27, 28]. Thicker EAT has also been associated with hemodynamic signs of pericardial restraint, supporting a potential mechanical contribution to impaired filling [28].

CMR provides a quantitative measure of this cardiac fat burden. In a cohort of patients with heart failure, higher CMR-derived EAT volume was associated with mortality and heart-failure hospitalization [29]. For imagers, measuring EAT therefore provides additional information regarding disease severity and prognosis, complementing the assessment of chamber size and function and of myocardial tissue.

EAT is also responsive to metabolic treatment in heart failure. In a pooled analysis of two randomized trials of patients with type 2 diabetes and heart failure, dapagliflozin reduced CMR-measured EAT area, independent of changes in body mass index [30]. This provides one of the first



2 Overview of three representative patients with different cardiovascular conditions and their respective epicardial adipose tissue (EAT) burden on CMR. For each patient, long- and short-axis views are shown; EAT is measured for the left and right ventricle separately using the right ventricular insertion points as limits. While the patients with aortic valve stenosis and HFpEF show a relatively high EAT burden, the patient with myocardial infarction only exhibits a thin EAT layer that separates from a small pericardial effusion (PE). These findings are only given as individual patient examples and do not represent differences in EAT burden across disease spectrums.
HFpEF = heart failure with preserved ejection fraction

concrete examples of CMR detecting treatment-associated changes in cardiac adiposity. However, it does not establish that EAT reduction itself mediates clinical benefit, nor does it justify using EAT measurements to initiate or adjust treatment.

2.4 Valvular heart disease

In aortic stenosis, EAT has attracted interest because of its association with myocardial remodeling and clinical outcomes [31]. In patients with asymptomatic aortic stenosis, larger EAT volumes were associated with greater left ventricular mass, larger left atrial volumes, impaired ventricular function, and higher troponin concentrations, while showing no association with stenosis severity or progression. Higher EAT volume was also associated with mortality after adjustment for age, body mass index, and left ventricular ejection fraction [32].

CMR allows this relationship to be examined within a single study by combining EAT volumetry with measurements of ventricular mass, myocardial fibrosis, and atrial function. In severe aortic stenosis, CMR-derived EAT volume has been independently associated with impaired left atrial reservoir strain [33], providing a specific link between cardiac fat accumulation and atrial dysfunction.

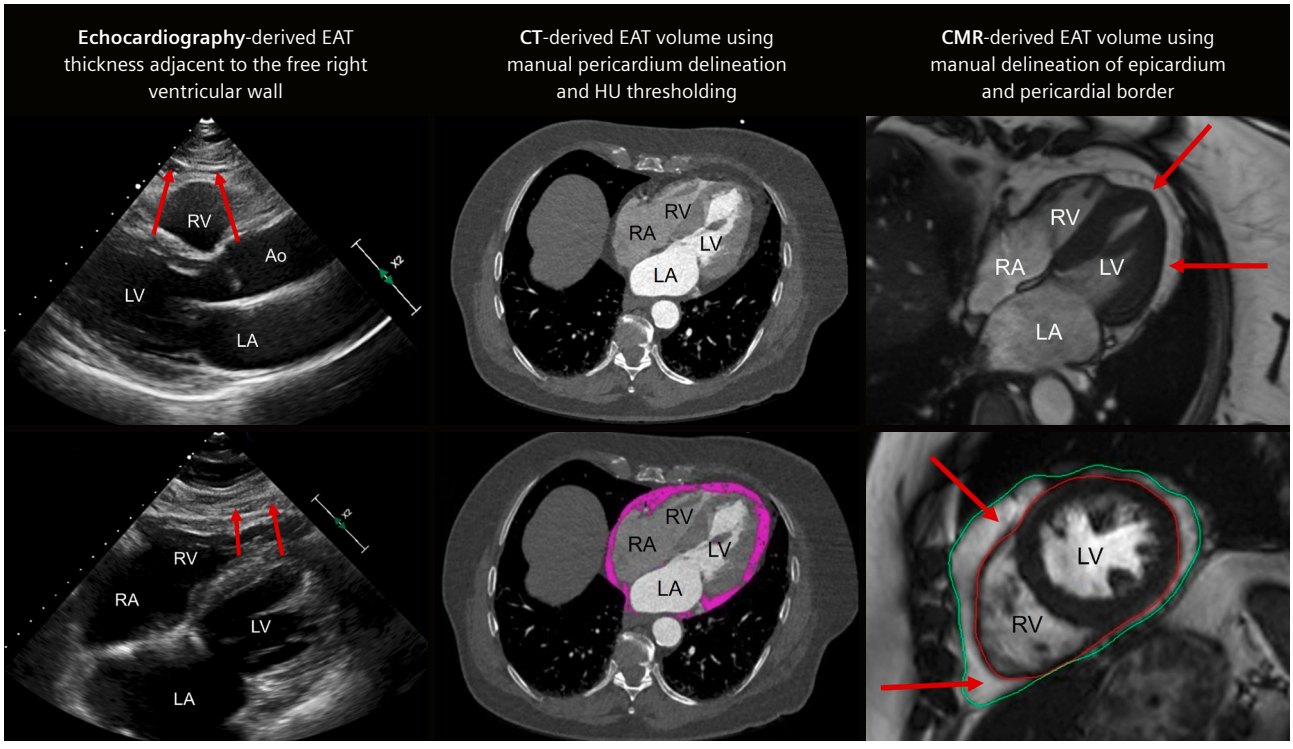
Assessing EAT alongside these established CMR parameters adds information about cardiac adiposity to the characterization of myocardial and atrial remodeling. Whether EAT directly contributes to these abnormalities, however, remains unresolved.

3. How to measure EAT with CMR

EAT can be assessed by echocardiography, CT, and CMR, but the measurement results are not interchangeable. Spatial resolution, tissue contrast, coverage, slice thickness, and anatomical definitions vary across modalities and studies. Standardized delineation is therefore essential, particularly at the pericardial boundary and the basal and apical limits. For maximum interpretability, acquisition and segmentation procedures should follow explicit and ideally standardized operating procedures.

3.1 Anatomical definition

Reliable measurement begins at the pericardium. The inner contour follows the epicardial surface; the outer contour follows the visceral pericardium, excluding paracardial fat outside the parietal pericardium. This thin boundary can be obscured by partial volume effects, especially at the



3 Comparison of epicardial adipose tissue assessment across modalities.
Ao = Aorta; CMR = cardiovascular magnetic resonance imaging; CT = computed tomography; HU = Hounsfield units; LA = left atrium; LV = left ventricle; RA = right atrium; RV = right ventricle

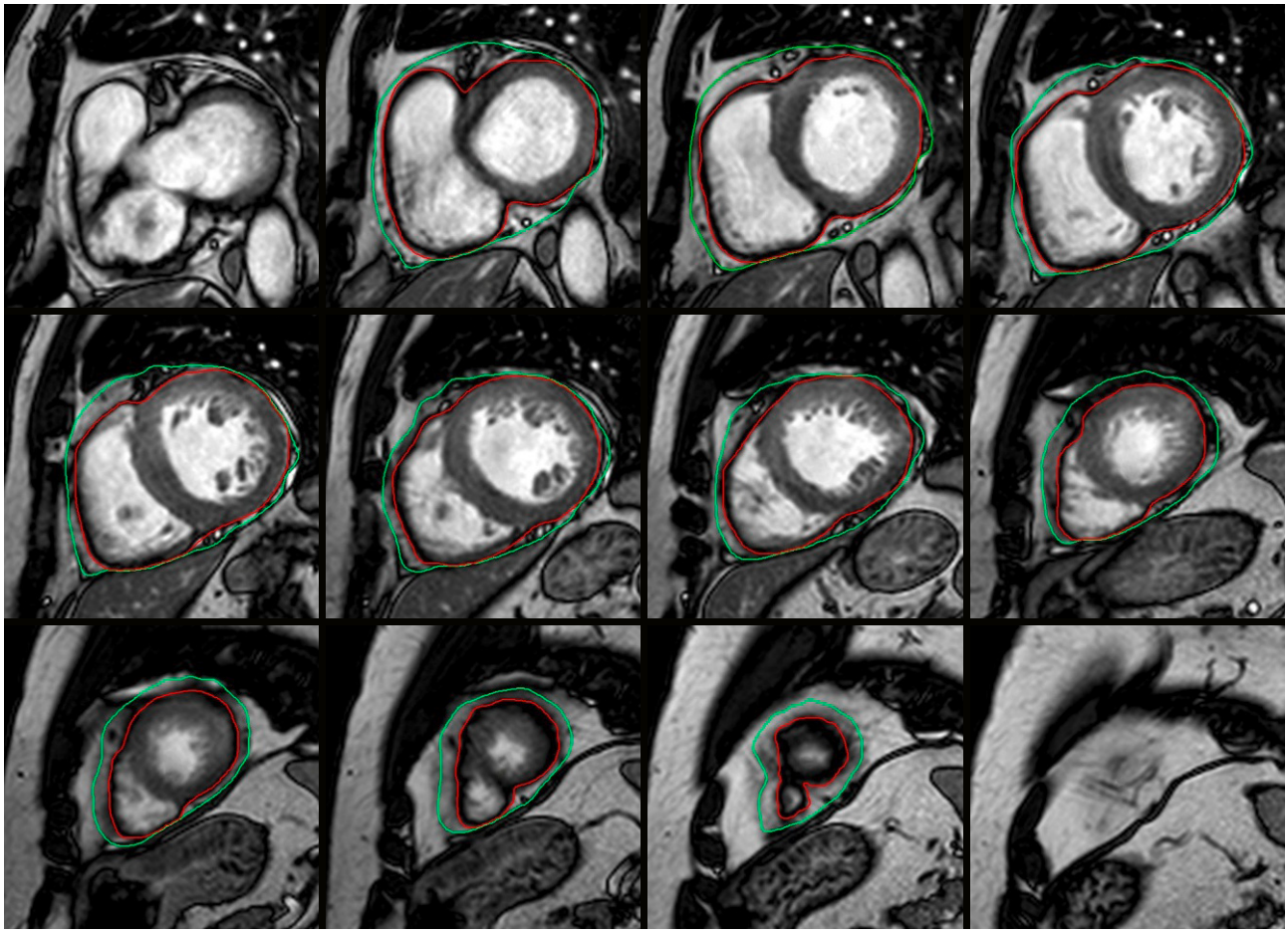
cardiac base, around the atria, and near the great vessels. Protocols should thus explicitly define basal and apical endpoints and apply them consistently.

3.2 Thickness, area, volume — and distribution

As illustrated in Figure 3, each imaging modality offers a different view of EAT and has its own strengths and limitations. Assessing EAT on images acquired as part of clinical routine provides a pragmatic starting point. Echocardiography offers a one-dimensional thickness measurement, which may not accurately reflect the total amount of fat surrounding the heart. Volumetric assessment captures EAT's uneven distribution more comprehensively and has become the mainstay for EAT quantification. Cardiac CT has been considered the reference standard due to its high spatial resolution and ability to identify EAT through the characteristic attenuation. However, the radiation exposure restricts the appeal of performing dedicated CT examinations solely for assessing EAT.

CMR allows EAT assessment alongside ventricular and atrial function and myocardial tissue characterization without ionizing radiation. While area measurements on individual images depend on the selected plane and may miss substantial fat elsewhere, contouring EAT across a stack covering the entire heart provides a more complete assessment of its volume and distribution. However, manual analysis is time-consuming, making automated segmentation an attractive approach for faster assessment, with visual review still required to ensure accuracy.

Standardized cine-derived CMR volumetry (Fig. 4) has demonstrated reproducibility, scan-rescan repeatability, and agreement with cardiac CT [34]. Nevertheless, differences in spatial resolution, slice spacing, coverage, and segmentation criteria can produce systematic differences between measurements. Comparisons should therefore use consistent protocols, and thresholds should not be transferred between methods without validation.



4 CMR-derived quantification of epicardial adipose tissue using a short-axis balanced steady-state free precession cine sequence including the most basal and apical slices without delineation. The green line follows the parietal layer of the serous pericardial border, and the red line marks the epicardium. Figure published in [34].

For transparent reporting, EAT volume should be provided in milliliters together with any indexed value. Different indexing schemes have been reported. Indexing to body surface area accounts for body size but can reduce apparent differences associated with obesity. Indexing to myocardial mass or myocardial volumes describes different relationships. In summary, the reference structure for normalization should be reported in addition to specifying whether measurements represent total EAT or a regional depot, such as fat surrounding the atria, ventricles, or coronary arteries.

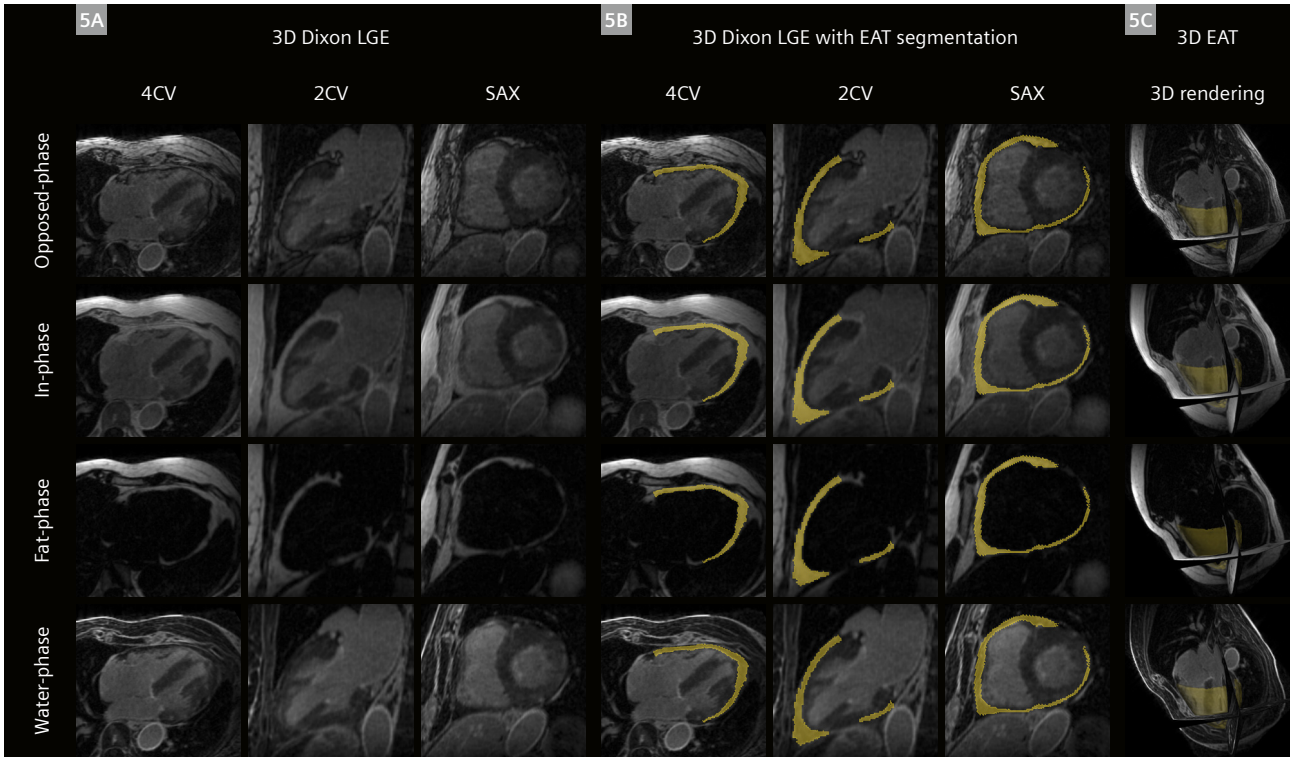
3.3 Sequences and technical choices

With CMR, EAT assessment can be readily incorporated into routine practice by leveraging short-axis cine images already acquired during standard CMR examinations. On balanced steady-state free precession images, fat typically appears bright, and the ventricular stack acquired in most clinical examinations allows retrospective analysis without additional scan time, provided coverage is sufficient. Although the thin pericardium can be difficult to identify on a single frame, reviewing the cine loop can help delineate its boundary through the relative motion of the heart with the adjacent EAT in comparison to the surrounding tissues during the cardiac cycle. Distinguishing EAT from pericardial fluid may nevertheless be challenging, and bright blood and artifacts can also obscure tissue boundaries. Thick slices introduce partial volume effects, and inter-slice gaps leave tissue unsampled. Consistent coverage and cross-checking in orthogonal planes support reliable segmentation.

4. Beyond volume: Qualitative characterization of EAT

4.1. 3D Dixon-based water-fat separation

In addition to conventional 2D Dixon techniques resolving fat and water signals, modern 3D Dixon techniques allow for direct tissue decomposition of the whole heart, clearly differentiating EAT from surrounding non-fat tissues (Fig. 5). For instance, in the case of pericardial effusion, the method's water-suppressed fat-phase allows for a precise evaluation of EAT that would otherwise be difficult to differentiate [35]. In particular, whole-heart 3D Dixon imaging addresses several physical constraints inherent to conventional 2D acquisitions: Providing isotropic, high-resolution cardiac coverage (such as 1.3 × 1.3 × 1.3 mm^3) eliminates slice gaps and drastically mitigates boundary-



5 EAT segmentation using a 3D Dixon approach. **(5A)** By utilizing in-phase and opposed-phase imaging, the 3D Dixon technique generates distinct fat-only and fat-suppressed (water-phase) images. **(5B)** These enable the precise manual or semi-automated delineation and accurate quantification of EAT. **(5C)** The isotropic resolution of the 3D sequence facilitates seamless multiplanar reformatting during postprocessing.

definition challenges in the delineation of thin EAT areas on 2D acquisitions. From a postprocessing perspective, an additional distinct technical advantage of 3D isotropic datasets lies in the capability for retrospective multiplanar reformatting in any arbitrary oblique plane. This enables cross-referencing of anatomical boundaries across multiple views and permits precise, targeted assessment of challenging local depots, such as fat surrounding the atria, the coronary arteries, or the ventricular borders [36]. Furthermore, combining Dixon principles with late gadolinium enhancement (LGE) imaging enables simultaneous discrimination between myocardial fibrosis and intramyocardial fat infiltration. By separating fat and water signals, Dixon-LGE thus prevents fat from mimicking or obscuring scar tissue in EAT-adjacent myocardium, providing additional diagnostic support in conditions characterized by fibrofatty myocardial remodeling [37]. However, one of the primary bottlenecks for the adoption of isotropic 3D Dixon acquisitions for quantitative EAT evaluation lies in the postprocessing workload: Manual slice-by-slice contouring across 80 to 100 contiguous slices may require up to one hour per case, rendering manual workflows impractical for clinical routine and large cohort analyses. Emerging automated deep-learning approaches overcome this limitation by operating directly on multi-channel Dixon images (opposed-phase, in-phase, fat-phase, and water-phase). By cross-referencing signal characteristics across all four phases simultaneously, the deep-learning models separate EAT from adjacent myocardium even in challenging scenarios such as myocardial scar, intramyocardial fat infiltration, or pericardial fluid, reducing postprocessing times from one hour per case to minutes, with reproducibility approaching human interobserver levels [38]. This also makes the technique attractive for large-scale cohort analyses, provided strategies for manual quality control are incorporated [39]. Furthermore, it paves the way for a more widespread adoption of EAT as a cardiometabolic imaging biomarker in both research and clinical routine.

4.2. Why EAT volume alone may be insufficient

While EAT volume has consistently been associated with adverse cardiovascular outcomes and structural remodeling, relying on volume alone presents clear limitations. Larger EAT burden does not necessarily have to act biologically equivalent across individuals. Two patients presenting with identical volumetric measurements may exhibit fundamentally disparate metabolic and clinical trajectories. While one patient's EAT depot might consist of relatively quiescent adipose tissue, another's could propagate inflammation, structural remodeling of adjacent tissue, and an altered microscopic lipid composition. Emerging evidence indicates that the biological state of EAT — determined by comorbidities, microscopic lipid composition, and inflammation activity — determines the pathological impact on the adjacent myocardium and coronary vasculature through paracrine and vasocrine signaling pathways [4]. Characterizing tissue composition rather than mere volume is therefore of increasing interest to cardiometabolic CMR.

In this regard, several emerging CMR techniques could redefine the joint qualitative and functional interrogation of EAT. Advanced parametric mapping sequences, including joint T1- and T2-based characterization (such as 3D whole-heart bright-blood and black-blood imaging with co-registered T1/T2 myocardial tissue mapping), show promise in evaluating intrinsic relaxation properties that reflect structural integrity and tissue water content [40]. Hence, they could also be used for the assessment of epicardial fat compartments. Furthermore, regional 3D mapping could facilitate detailed topographic evaluation of EAT adjacent to the coronary arteries, atria, and ventricular myocardium, helping distinguish depot-specific mechanistic effects of EAT such as atrial remodeling [10] or pericoronary plaque destabilization [12]. Combined with a comprehensive functional examination, these emerging quantitative approaches extend EAT assessment from bulk volumetry into phenotype-specific cardiometabolic profiling.

5. Outlook

For EAT assessment to become practical in clinical care, analysis should require minimal additional effort while remaining transparent and easy to review. Automated segmentation could derive EAT volume from routine cine images or dedicated Dixon datasets and present global and regional measurements alongside ventricular volumes.

The next step is to place EAT quantity, distribution, and composition within the broader cardiac assessment. CMR is particularly well suited to this task because it combines fat assessment with stress perfusion, myocardial tissue differentiation, and ventricular and atrial function in a single examination. Regional EAT measurements could therefore be related to abnormalities in adjacent structures. This integrated approach offers a way to investigate how cardiac adiposity relates to the functional and structural changes observed in cardiovascular disease.

Clinical translation will require multicenter reference ranges; harmonization across field strengths, vendors, sequences, and reconstruction methods; and external validation in diverse populations. Most importantly, outcome-based thresholds must show incremental value beyond established clinical and CMR markers. EAT will become useful not when it can be measured, but when its measurement changes how a patient is understood, monitored, or treated.

6. Conclusion

Epicardial adipose tissue occupies a uniquely informative position at the interface of systemic metabolism and local cardiac disease. Its proximity to the coronary arteries, atria, and myocardium makes it biologically compelling, while its routine visibility makes it attractive as an imaging biomarker. CMR adds decisive advantages: EAT can be quantified without ionizing radiation and interpreted together with perfusion, function, fibrosis, and remodeling in a single examination.

Volumetric assessment is currently the most comprehensive quantitative measure, but its interpretation depends on standardized anatomical boundaries, cardiac coverage, and robust reproducibility. Three-dimensional and Dixon water-fat imaging may improve spatial delineation and add information on fat composition, while automated segmentation can make volumetry scalable. These developments may transform EAT from a structure that is routinely seen but usually ignored into a component of cardiometabolic CMR phenotyping. The remaining challenge is not simply to measure EAT faster, but to determine when its quantity, distribution, or quality provides clinically actionable information beyond established risk factors and myocardial imaging markers.

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Contact

Judith Gronwald, M.D.
Department of Cardiology and Pneumology
University Medical Center Göttingen
Georg-August University Göttingen
Robert-Koch-Strasse 40
37075 Göttingen
Germany
Tel.: +49 551 399625
judith.gronwald@med.uni-goettingen.de

