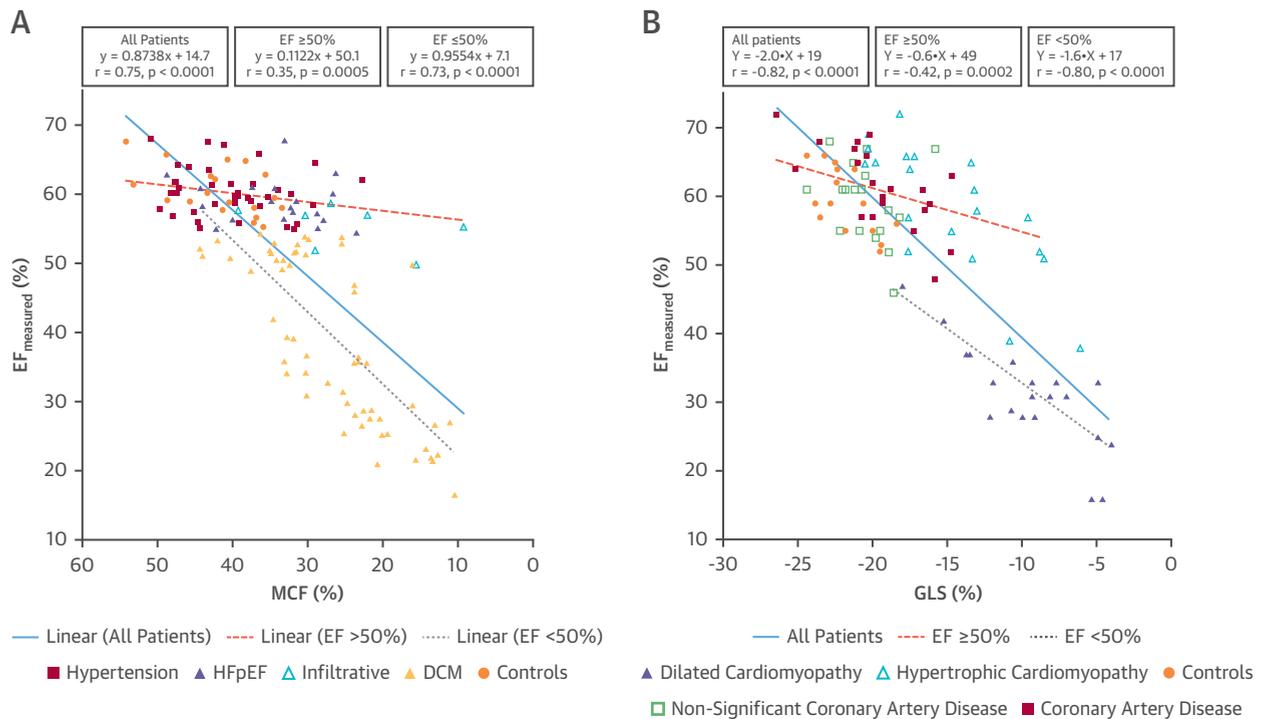


FIGURE 1 Correlation Between MCF, GLS, and EF

(A) Correlation of myocardial contraction fraction (MCF) with ejection fraction (EF) by using freehand 3-dimensional echocardiography. There were 20 control subjects, 39 subjects with hypertension, 59 subjects with dilated cardiomyopathy (DCM), 8 subjects with infiltrative diseases, and 24 subjects with heart failure with preserved ejection fraction (HFpEF). **(B)** The regression analysis reveals a markedly weaker correlation between EF and MCF in the patient group with EF $\geq 50\%$ compared with the group with EF $< 50\%$, similar to what was reported by Stokke, et al. (1) for global longitudinal strain (GLS).

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Complexities in Modeling the Relationship Between Longitudinal Strain and Ejection Fraction



Stokke et al. (1) have performed a combined echocardiographic and mathematical modeling study to investigate the relationship between left ventricular (LV) ejection fraction (EF), global longitudinal strain (GLS), and global circumferential strain (GCS). On the basis of modeling using an equation for EF that included both GLS and GCS as independent variables, and that was validated against echocardiographic EF

in the population studied, the authors concluded that GCS contributed more than twice as much to EF compared with GLS. However, as recognized by the authors, this conclusion seems to be inconsistent with a previous cardiac magnetic resonance study by Carlsson et al. (2), which reported that long-axis LV contraction accounts for approximately 60% of the LV stroke volume. Understanding the reason for the apparent divergence between the findings and conclusions of Stokke et al. (1) and Carlsson et al. (2) is therefore of considerable importance.

A fundamental principle underlying the cardiac magnetic resonance study of Carlsson et al. (2) was that apically directed motion of the mitral annulus resulting from contraction of LV long-axis fibers, and therefore shortening of the left ventricle in its long axis, must also lead to short-axis (radial) thickening. This principle is based on conservation of LV mass, which still applies even if a small percentage of myocardial compressibility is assumed, as it was in the study of Stokke et al. (1). The only way that long-axis contraction-mediated short-axis thickening would not be contributing to a lower LV end-systolic volume, and thus larger stroke volume and absolute value of GCS, is if the epicardial border expanded at the same time. However, it is the opposite which occurs (3,4). Hence, an explanation for the apparent discrepancy in the findings of the 2 studies is an intrinsic limitation of the equation used for modeling the effects of changes in GLS on EF, as independence of GLS and GCS is assumed in the equation and a dependent change in GCS as a result of a change in GLS is not possible.

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REPLY: Interaction Between Longitudinal, Circumferential, and Radial Deformations and Their Contributions to Ejection Fraction



We read with interest the comments from Dr. Matthews and colleagues who propose "myocardial contraction fraction" (MCF) as an alternative systolic function parameter. We believe it is difficult to evaluate this parameter due to the limited number of studies applying MCF. However, MCF can easily be extracted in our mathematical model, and we encourage such a theoretical investigation, which we believe would provide valuable insights regarding the relation between MCF versus strains, ejection fraction (EF), wall thickness, and cavity volume.

We also appreciate the comments by Dr. Carlsson and colleagues, as the different assumptions we use in our respective models (1,2) and the consequently diverging conclusions should be made clear. The main difference in our assumptions is with respect to epicardial motion, as also pointed out by Dr. Peverill in his letter. We agree that there is no outward epicardial motion; rather, it moves slightly inward. As the longitudinally oriented fibers contract, they generate stretching forces in both the radial direction, giving rise to 1 component of wall thickening, and also in the circumferential direction. However, there is no circumferential stretch because there are actively contracting circumferentially oriented fibers. Active circumferential fiber shortening directly reduces the cavity diameter and indirectly reduces it further by generating another component of radial stretching forces that contribute to the wall thickening.

Hypothetically, a left ventricle with only longitudinal fibers would only have passive intercellular and intracellular tissue structures resisting the increased pressure during systole in the circumferential direction. Hence, these would stretch (due to increased pressure + Poisson's ratio effect), resulting in an outward motion of the epicardium. Similarly, in a hypothetical ventricle with only circumferential fibers, it would stretch in the longitudinal direction during systole. We have already performed a finite element simulation study that showed this effect (3).