Cardiac enlargement among trained athletes was first documented more than a century ago. Since the pioneering work of Henschen and Darling, both visionary investigators who independently and nearly simultaneously described enlarged cardiac silhouettes among Nordic skiers and rowers, respectively, much has been learned about the relationship between vigorous exercise and cardiac structure and function. Several decades of cross-sectional studies using multimodality noninvasive imaging techniques have established characteristic features of the athlete’s heart including balanced biventricular dilation, mild-to-moderate increases in left ventricular wall thickness, and biatrial dilation. More recently, carefully conducted longitudinal studies have established a cause and effect relationship between exercise training and cardiac remodeling. The term exercise-induced cardiac remodeling (EICR), now in widespread use, encompasses the many structural and functional adaptations that occur in response to routine exercise training. In clinical practice, findings consistent with EICR are the norm, not the exception, and should be viewed as adaptive physiology rather than subclinical disease among asymptomatic competitive athletes. It must, however, be emphasized that exercise, the stimulus for EICR, is not a binary factor but rather a continuous and complex variable that is defined by the cross product of intensity, frequency, and duration. In clinical practice, findings consistent with EICR are the norm, not the exception, and should be viewed as adaptive physiology rather than subclinical disease among asymptomatic competitive athletes. It must, however, be emphasized that exercise, the stimulus for EICR, is not a binary factor but rather a continuous and complex variable that is defined by the cross product of intensity, frequency, and duration. In between sedentary living, perhaps our greatest contemporary public health threat, and competitive athletics exists a broad exercise dose range. To date, the vast majority of previous EICR studies have examined elite or subelite athlete cohorts with individuals who exercise at a minimum of 10 hours per week and routinely include high-intensity efforts to prepare for competitions. As such, comparatively little is known about cardiac adaptation among the much larger segment of the general population that exercises at less extreme doses. To what degree EICR occurs among recreational exercisers and how EICR impacts relevant health outcomes remains largely unknown.

The opinions expressed in this article are not necessarily those of the editors or of the American Heart Association.

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dose-dependent manner. Second, the finding of indirect relationships between ventricular ejection fractions and exercise exposure emphasizes the critical importance of applying basic principles of physiology during imaging test interpretation. Resting cardiac output, the product of stroke volume and heart rate, is determined by basal metabolic needs, which fluctuate minimally with changes in physical fitness and exercise habits. Larger ventricles therefore require lower ejection fractions than smaller ventricles under resting conditions, a phenomenon that is often misinterpreted as evidence of cardiomyopathy in routine clinical practice. Finally, careful inspection of data contained in Table 2 suggests a clear exercise dose threshold for EICR. Specifically, participants in physical activity category II (i.e., those doing ≤3 hours of exercise per week) showed no statistical differences when compared with sedentary reference participants in category I. This suggests that people who exercise up to doses suggested by current US physical activity guidelines, recommendations with a firm epidemiological foundation geared toward optimizing health and longevity, should not be expected to demonstrate features of EICR. To what degree EICR independently impacts health outcomes among people who choose to exceed exercise dose recommendations, an increasing segment of the population that largely enjoys superb health and quality of life remains uncertain.

Having considered the advances afforded by data from the UK Digital Heart Project cardiac magnetic resonance project, we turn our attention to on-going areas of uncertainty. The suggested EICR dose threshold (i.e., routine exercise exceeding 3 hours per week) should be considered speculative and over simplistic. Self recall surveys that query exercise frequency and duration have imperfect accuracy but most importantly fail to account for exercise intensity. It is almost certain that the intensity of exercise, typically quantified as some percentage of peak effort, plays an important role in the process of EICR. Thus, it is likely that people who exercise intensely for relatively short periods of time may undergo EICR, whereas those who spend longer periods of time exercising at low-to-moderate intensity may fail to remodel. Future data collection techniques, many of which will rely on technological advances that enable real-time capture of exercise habits, will be required to develop a more informed and complete understanding of how each component of the exercise dose independently contributes to EICR. Next, beyond ejection fraction, the present data set tells us little about the functional attributes of EICR among moderate exercisers. Previous work suggests that lifelong competitive athletes seem largely spared from the typical declines in left ventricular diastolic function that accompany advancing age, a finding that is expected to confer an adaptive advantage in later life. How much exercise is required to avoid potentially deleterious age-associated changes in ventricular compliance remains unknown and constitutes an area of important previous work. Finally, the current study, largely based on the decision to forgo contrast-enhanced cardiac magnetic resonance, provides no information about cardiac fibrosis among moderate exercisers. Conflicting reports about the prevalence of nonischemic ventricular fibrosis among high-end athletes, a finding that has been suggested as a form of overuse cardiac pathology, has yet to be rigorously assessed in large studies with cohorts covering the entire exercise dose spectrum. As such, we remain unable to determine whether excessive EICR has the potential to result in a malignant phenotype and if so why.

Cross-sectional data presented by Dawes et al represent an important step in our quest to understand cardiac remodeling. Yet, they simultaneously remind us of a larger overarching principle in clinical imaging. Clinicians who sit in dark imaging suites making careful quantitative assessments of cardiac structure and function will best serve their patients and colleagues by vigilantly remembering that no image should be divorced from the person from whom it was generated. Determining the clinical significance of cardiac chamber dilation and hypertrophy is of limited value unless key information about both the patient and the reason for the imaging test are readily available. In real time, this can easily be accomplished by taking the time to discuss testing indications with ordering providers to ensure that the findings either do or do not meet a priori expectations. Future expansion and refinement of electronic medical record systems may facilitate this process but nothing should ever replace the thoughtful conversations between cardiac imagers and clinicians in the trenches that lead to optimal patient care.

Disclosures
None.

References


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Exercise-Induced Cardiac Remodeling: Competitive Athletes Are Just the Tip of the Iceberg
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