The Heart of a Champion*

Paul S. Bhella, MD, Benjamin D. Levine, MD

Dallas, Texas

Elite endurance athletes are the paragons of circulatory health—able to generate extremely high rates of oxygen uptake by virtue of “Olympic sized” oxygen transport systems. The primary cardiovascular adaptation of an endurance athlete that allows for such extraordinary aerobic power is a very large stroke volume (1) achieved by a large, compliant heart (2) that relaxes quickly (3). Although these changes facilitate an augmentation of cardiac output in response to increased metabolic demand, some have questioned whether they might be accompanied by yet unrecognized, long-term risks. Indeed, the term “the athlete’s heart” was originally coined to suggest pathological cardiac enlargement as perceived by early clinical investigators (4). Supporting this notion are the well-described phenomena of “cardiac fatigue” (5), post-exercise elevations in cardiac specific biomarkers (6), and long-term changes in cardiac morphology that might mirror those observed in pathologic conditions. In this issue of the Journal, Pelliccia et al. (7), who have contributed much to this field, further explore this risk/benefit relationship by reporting the results of a longitudinal cardiovascular evaluation in 114 highly successful and intensively trained Olympic endurance athletes.

“Cardiac fatigue,” a phrase coined to describe transient changes in left ventricular performance after extraordinary endurance events, has been observed by several investigators after a variety of athletic endeavors. Douglas et al. (5) brought these observations to the mainstream in the late 1980s in their study of 21 athletes at the Hawaii Ironman Triathlon, an event lasting approximately 8 to 9 h for the fastest runners. With echocardiography before and after the race and after a period of recovery, the authors observed a reduction in fractional shortening by 10% from pre-race to post-race studies, only to have this index return to pre-race levels in the recovery period (5), a finding that has been confirmed by these and other investigators (8). Elevations in cardiac-specific biomarkers, such as troponin I and T, have also been noted after endurance events; although in contrast to the cardiac fatigue studies, these elevations seem to be greatest in the least trained and slowest runners (9). Used predominantly in the diagnosis of acute coronary syndromes, these biomarkers are highly sensitive for myocardial injury (or at least changes in membrane permeability), with elevations also noted in myocarditis, pulmonary embolism, and heart failure. In a recent meta-analysis by Shave et al. (6), pooling 1,120 endurance athletes, post-exercise troponin T exceeded the lower limit of detection (0.01 μg/l) in just under one-half of the participants, revealing the high prevalence of mild elevations of troponin in this population. Finally, the typical description of the athlete’s heart, one which features an enlarged left ventricular chamber with thickened walls and a resting ejection fraction at the lower limits of normal, often mirrors some changes observed in pathological conditions such as hypertrophic or dilated cardiomyopathies. Hence, although specific parameters for differentiating the athlete’s heart from pathophysiologic responses to cardiovascular disease have been defined (10) and prove helpful in the majority of cases, some fraction of athletic hearts will exceed these cutoffs, especially in large male athletes like the rowers and canoeists studied in this paper. Whether the cardiac phenotype of these athletes represents an extreme form of benign remodeling or rather reflects pre-clinical pathology is a matter of some uncertainty.

Although the mechanisms that underlie these divergent observations are poorly understood, the question of whether repetitive episodes of subtle injury might lead to long-term changes in left ventricular morphology and function is germane. Recurrent subtle injury might lead to alterations of systolic or diastolic function, as has been suggested in cyclists who have competed in multiple Tour de France races (11) or older marathon runners competing in multiple marathons (12).

To address this question, Pelliccia et al. (7) used physical examinations, electrocardiograms, and echocardiograms performed by the Italian National Olympic Committee and reported the results of serial examinations of endurance athletes who competed in a minimum of 2 Olympics games, with a mean follow-up of 9 years. Key findings included: 1) the absence of clinical events including syncope, cardiac arrest, or sudden cardiac death; 2) the absence of progressive changes in left ventricular morphology, including end-diastolic volume, end-systolic volume, and wall thickness; and 3) the absence of changes in contractile function, as assessed by ejection fraction and the ratio of peak systolic pressure to end systolic volume (PSP/ESV).

The work of Pelliccia et al. (7) is to be commended, because it offers objective evidence that prolonged and recurring Olympic level training requiring high volume and intensity of training as well as repetitive competitions does

*Editorials published in the Journal of the American College of Cardiology reflect the views of the authors and do not necessarily represent the views of JACC or the American College of Cardiology.

From the Institute for Exercise and Environmental Medicine, Texas Health Presbyterian Dallas, and the Division of Cardiology, University of Texas Southwestern Medical Center, Dallas, Texas.
little to alter cardiac phenotype or performance. The addition of a control group with persistent training but failure to qualify for a second Olympic Games is a further strength and gives confidence that the results are not exclusively due to selection bias where only the athletes with preserved function were able to remain successful at this level. Moreover, these Olympic athletes were carefully monitored and screened for doping, a problem that has plagued competitors in the Tour de France and that might have confounded previous research in this area (11). Lastly, as noted by the authors, virtually all studied athletes were Italians and Caucasian; athletes of African origin might have measurable differences in cardiac morphology (13). Nonetheless, the high internal validity of this population makes up for any limitation in external validity, although additional studies in non-Caucasian athletes are warranted.

In contrast to the study’s numerous strengths, a few limitations should also be considered. Most evident is that subtle changes in left ventricular systolic or diastolic function might escape detection of crude measures of systolic performance such as the ejection fraction. Although a cornerstone of clinical echocardiography, this index is exquisitely load-dependent and prone to high test-retest variability. Fortunately, the addition of an ejection phase index normalized to afterload, such as the peak systolic pressure/end-systolic volume provides additional confidence that systolic function remained normal in this population. Moreover, in the authors’ defense, all echocardiographic studies were performed before 2005; since then, more sensitive techniques have become available, including myocardial tissue Doppler, strain analysis, and strain rate analysis. Subtle changes in cardiac function over shorter periods of time might require more sophisticated, sensitive techniques such as these.

The development of myocardial fibrosis and consequent risk of arrhythmias is a final topic of concern for the athlete’s heart. Whether years of training and competition might lead to a fibrotic heart prone to arrhythmia is arguable (12), although until now there were virtually no longitudinal studies examining this issue. Such is the scenario posed in a case report by Whyte et al. (14), who detail the case of a 57-year-old competitive marathoner who died while running; postmortem evaluation revealed a hypertrophic and fibrotic heart, leading the authors to speculate these findings were secondary to years of extensive training and competition. The presence of delayed enhancement of gadolinium on cardiac magnetic resonance imaging (cMRI) in approximately 5% of older marathon runners in a nonischemic pattern is supportive of this hypothesis (12), although this frequency was not clearly distinguishable from that observed in a control population of similar age (50 to 72 years). Moreover, it is impossible to separate the effects of marathon running itself from the effects of other clinical considerations, such as intensive exercise training in the presence of subclinical coronary heart disease in such patients. In light of these concerns, the absence of syncope, cardiac arrest, or sudden cardiac death in the Italian cohort is certainly reassuring, as is the lack of change in cardiac morphology. It should be noted, however, that 2 of these 114 athletes did indeed have significant ventricular arrhythmias that required medical intervention, so this issue cannot be completely ignored, especially given a preliminary, unpublished report that suggests that even some younger athletes might have increased delayed enhancement on cMRI (15).

The data now reported by Pellicia et al. (7) in young elite competitive athletes in a longitudinal fashion are scientifically more rigorous to answer the question at hand—does prolonged, intensive exercise by itself injure the heart? Whether another 20 years of marathon-style training is sufficient to cause myocardial injury is possible, although it is not clear why that should be the case, given the remarkable stability of cardiovascular structure and function over nearly 1 decade of elite training and competition. Moreover, it is reassuring that recent studies show no evidence of increased delayed enhancement in the myocardium after a marathon, despite elevations in plasma biomarkers (16). It must be acknowledged, however, that the presence of arrhythmogenic foci and subclinical episodes of arrhythmia were not thoroughly assessed in the work by Pellicia et al. (7). Use of advanced tools such as cMRI might be necessary to apply on a systematic basis, longitudinally in both young and older athletes, to convincingly disprove this hypothesis.

The athlete’s heart remains as much a mystery as a marvel. Whether observations such as cardiac fatigue, elevated biomarkers, and pronounced morphologic and functional changes represent benign physiologic adaptations or cross into the category of pathology remains uncertain. However, the report by Pellicia et al. (7) goes a long way to reassure the medical community of the inherently physiologic and clinically benign nature of prolonged and intense endurance training.

Reprint requests and correspondence: Dr. Benjamin D. Levine, Institute for Exercise and Environmental Medicine, 7232 Greenville Avenue, Suite 435, Dallas, Texas 75231. E-mail: BenjaminLevineMD@texashealth.org.

REFERENCES


Key Words: intense athletic training • left ventricular function • long-term follow-up • Olympic athletes.