EDITORIAL COMMENT

Citius, Altius, Fortius
(The Olympic Motto: Swifter, Higher, Stronger)*

Pamela S. Douglas, MD, FACC
Madison, Wisconsin

In this Olympic year, as since time immemorial, we are fascinated with the achievements of athletes. As cardiologists, we have a special interest in the athlete’s heart, how it adapts to exercise training, what its limits are, and whether exercise can cause harm. In the 1950s, Paul Dudley White performed an autopsy on Clarence DeMar, a perennial Boston Marathon winner, and noted an enlarged heart and arteries. As new imaging techniques such as echocardiography have confirmed that cardiac structural changes are an integral part of the response to exercise, investigators and clinicians alike have struggled to differentiate the physiologic effects of exercise training from the pathologic entities of hypertrophic and dilated cardiomyopathy.

A separate but related area of investigation seeks to define the upper limits of cardiac adaptation to the physiologic stimulus of exercise. Recently, a genetic component to physiologic hypertrophy has been confirmed, revealing that the “gain of function” model of the athlete’s heart is influenced by genetics as well as environmental factors such as the type and duration of training (1,2).

See page 144

In this issue of the Journal, Abergel et al. (3) shed light on both these concerns through an echocardiographic study of a large number of elite professional cyclists participating in the Tour de France. They noted marked left ventricular (LV) dilation in a subset of athletes, with more modest increases in wall thickness. Their findings confirm previous studies of Olympians (4), ultradistance triathletes (5), and others: endurance athletes often have substantial cardiac dilation coupled with modestly increased wall thickness. Although cardiac remodeling lies within the range of normal for most athletes, it results in mild eccentric hypertrophy in many.

Although Abergel et al. (3) have carefully used a contemporaneous control group to define the normal ranges of LV dimensions and mass, for the important entity of eccentric remodeling, they use a single cut point (0.44), whose derivation is unreferenced. As a cut point, 0.44 defines the limit of concentric, not eccentric, remodeling. In fact, the mean value for relative wall thickness in controls is 0.342, whereas that in athletes is actually higher (less eccentric geometry) at 0.354 (p = 0.06). Clearly the definition of “normal” will dramatically alter results; the use of the Abergel et al. (3) controls suggests far less eccentric remodeling than is claimed. Nevertheless, cavity diameter was increased to a more marked degree than has previously been reported.

Among the influences creating the structural adaptations in athlete’s heart, foremost is undoubtedly the physical training itself. In this respect, cycling is a combination of aerobic and resistance training. Even among cyclists, training for the Tour de France is notable for its endurance and, hence, the sheer volume of training required. Furthermore, of 29 sports studied in 1,309 Olympic athletes (6), cycling had the most marked impact on athletic “remodeling.” Thus, the Abergel et al. (3) findings can justifiably be considered to identify the upper limits of normal of cardiac exercise adaptation in any sport.

Other environmental factors may affect cardiac adaptation to exercise, including the overall duration of training, phase of training, hydration, time since last exercise bout, and so on. A prominent concern is the use of performance-enhancing drugs, including but not limited to anabolic steroids, growth hormones, erythropoietin, and stimulants—which are notoriously prevalent in the cycling world and whose long-term cardiac effects are incompletely studied (7). The impact of such substances upon the findings of Abergel et al. (3) cannot be known, but they may have contributed to the more extreme findings than previously reported. Furthermore, increased use over time may have been responsible for the more marked changes in the 1998 cohort.

Regardless of the causal stimuli, much has been written about differentiating athlete’s heart from hypertrophic cardiomyopathy (HCM) (8). Discriminating features include family and athletic history, cavity size, magnitude and asymmetry of hypertrophy (no athletes had a wall thickness >15 mm or septal/posterior wall ratio of >1.5) and, perhaps most distinctively, diastolic function. The importance and power of diastolic function measurement in screening for HCM was highlighted recently when tissue Doppler was used successfully to identify HCM carriers even in the absence of any other evidence of pathologic phenotype, including any increase in wall thickness (9).

In addition to differentiation from concomitant pathologic hypertrophy, the magnitude of LV dilation observed by Abergel et al. (3) overlaps with that observed in cardiomyopathy. Of even greater concern is the reduced ejection fraction noted in 12% of athletes with extreme cavity dilation, although most of these were not unexpectedly low, given calculated afterload. Even if asymptomatic cardiomyopathy can be excluded, similar degrees of dilation predict future development of cardiomyopathy, even in the absence of ischemic syndromes (10). Although less attention has been paid to the differentiation of an athlete’s heart from an early stage of dilated cardiomyopathy than from HCM (perhaps because of the clear relationship between sudden
death and exertion in this disease), LV dilation is probably no less relevant a target for preparticipation screening than wall thickness. This is also an important area for future research. Most studies of the athletic heart are cross-sectional—few provide serial data. In this respect, the more marked LV dilation observed by Abergel et al. (3) in the same athletes in 1998 compared with three years earlier is of substantial interest. It is possible that some of the training or environmental stimuli to physiologic hypertrophy were more marked, but the issue of possible harm from high-intensity, prolonged exercise training and competition is raised. In this respect, even single episodes of prolonged exercise result in transient decreases in LV systolic function, along with low-level troponin leakage, suggesting that myocardial injury can result from exercise (11,12). It is easy to imagine that repeated “minor injuries” of this type might eventually cause significant pathology—perhaps in the form of the excessive chamber dilation noted by Abergel et al. (3). Although it is of course beyond the scope of the Abergel et al. paper (3) to identify any causal relationship, it is hard to ignore the possibility that extreme amounts of exercise may cause harm. Fortunately, because few individuals train and compete at the extraordinary level of Tour de France cyclists, its relevance is probably limited. It appears that, even with exercise, one of the most powerful strategies for good health and longevity is moderation.

Reprint requests and correspondence: Dr. Pamela S. Douglas, Section of Cardiovascular Medicine, University of Wisconsin, H6/352 (3248) Clinical Science Center, 600 Highland Avenue, Madison, Wisconsin 53792. E-mail: psd@medicine.wisc.edu.

REFERENCES