

Editorial



Clinical Implications of Changes in Cardiac Structure and Function after Extreme Endurance Exercise

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Conflict of Interest

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Cardiac adaptations related to exercise were first documented in 1899. After the most intense exercise, the heart was described as being enlarged upon physical examination using auscultation and percussion.¹⁾ Later studies using electrocardiography, chest radiography, and echocardiography identified cardiac structural and functional changes caused by exercise.²⁾³⁾ It is important to distinguish between acute and chronic cardiac adaptation responses when confirming changes in cardiac structure and function induced by exercise. It has been shown that chronic adaptation in an endurance-trained athlete includes increases in left ventricular inner diameter and volume,⁴⁾ and exercise also has been shown to decrease cardiac contractility and ventricular relaxation as acute responses immediately following high-intensity endurance training.⁵⁾⁶⁾ During exercise, there is a need to shift the distribution of cardiac output into the skeletal muscles; heart rate and stroke volume increase during intense levels of exercise, which result in increased cardiac output.⁷⁾ Corresponding with these change, an immense increase in blood flow raises blood pressure (BP), and this peak exercise-related BP may play an important role in the development of ventricular hypertrophy. However, the exact mechanism for acute and chronic cardiac function and structure responses to endurance exercise has not yet been clarified. In addition, few studies have described the degree of cardiac structural and functional change before and after endurance exercise in detail using echocardiography.

In this issue of Journal of Cardiovascular Imaging, Kim et al.⁸⁾ assessed the changes in cardiac structure and function in participants performing a six-day super-ultramarathon (622 km). They revealed cardiac deformities and resilience seen as acute cardiac physiologic changes after long distance/long time endurance exercise. Among fifteen participants who were finally enrolled, left ventricular mass and mass index significantly increased after a super-ultramarathon compared to before the marathon; left ventricular ejection fraction and stroke volume were significantly decreased. Only half of the participants had echocardiographic data at the time of recovery, which showed that cardiac structure and function were significantly recovered compared with the time immediately post-marathon. Thus, they demonstrated that running and walking over 100 km/day for 6 days immediately increased left ventricular mass by more than 30%, which was accompanied by decreased cardiac function, which proved to be temporary and was recovered within a week. Although this study was limited by its small numbers of participants, its advantages over other studies is

that participants performed endurance exercise over a very long distance for a long period of time and results showed changes in cardiac variables before, and immediately after exercise, and after a subsequent recovery period. The exact mechanisms for acute responses in cardiac function and structure to endurance exercise was not investigated in this study, but its results provide evidence for acute responses in cardiac adaptation to endurance exercise. In the future, there will be many subsequent studies based on this study, i.e., it would be interesting to evaluate changes in cardiac structure and function by running distance and running time for a larger number of participants.

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