A Modern Definition of the Athlete's Heart—for Research and the Clinic

Rhys Beaudry, BSc[a,b], Mark J. Haykowsky, PhD, MSc[a,b], Aaron Baggish, MD[c], André La Gerche, MBBS, PhD, FRACP, FESC[a,d,*]

KEYWORDS

• Athlete • Heart • Cardiac • Remodeling • Hypertrophy

KEY POINTS

- There is need for a standardized framework to define the association between exercise training and the resulting changes in cardiac structure.
- There is a strong relationship between training amount (both intensity and duration), resulting fitness (often measured as Vo_{2max}) and cardiac size (volumes and mass).
- Additional factors including race, gender, and age have important influences on cardiac size.
- The relationship between training, fitness, and cardiac size can be exploited to determine whether cardiac changes are physiologically appropriate or suggestive of pathology.
- Estimates of training load and quantification of exercise conditioning are critically important factors necessary for understanding cardiac adaptation in research settings.

INTRODUCTION

Exercise can have a profound effect on cardiac size, mass, structure, and function, often referred to as the athlete's heart.[1] A consistent definition of what constitutes an athlete is essential for enabling researchers and clinicians to understand the considerable heterogeneity that exists in the literature regarding the degree of cardiac changes that may be expected as a result of exercise training. A diagnostic grey zone between athlete's heart and inherited cardiomyopathies exists at the extremes of athletic cardiac remodeling, and questions arise as to whether the observed increase in cardiac dimensions is proportional to the amount of exercise conditioning. This has significant implications. If the cardiac remodeling is consistent with that expected for the level of training, then further investigations may not be required. Conversely, if the remodeling is greater than might be expected, then it may imply an inherited cardiomyopathy. However, it can be extremely difficult to estimate the degree of expected remodeling given the broad ranges published in the literature. For example, cardiac mass has been reported to be anywhere between 9% and 90% greater in athletes as compared with nonathletes.[2] Part of this variance may be explained by differences in measuring cardiac dimensions, but much is also likely due to differences in athletic conditioning of the cohorts encompassed under the term athlete. Current

Disclosures: None.

[a] Department of Sports Cardiology, Baker IDI Heart and Diabetes Institute, 99 Commercial Road, Melbourne 3004, Australia; [b] College of Nursing and Health Innovation, University of Texas at Arlington, South Nedderman Drive, Arlington, TX 76019, USA; [c] Division of Cardiology, Massachusetts General Hospital, 55 Fruit Street, Boston, MA 02114, USA; [d] Department of Cardiology, Alfred Hospital, Melbourne, Australia

* Corresponding author. Sports Cardiology, Baker IDI Heart and Diabetes Institute, Level4 Alfred Centre, 99 Commercial Road, Melbourne 3004, Australia.

E-mail address: Andre.LaGerche@bakeridi.edu.au

Cardiol Clin ■ (2016) ■–■

http://dx.doi.org/10.1016/j.ccl.2016.06.001

guidelines that attempt to define athletic status use sport-specific classifications and use exercise hemodynamics as a means of explaining the observed differences in cardiac remodeling.[3] This article discusses the strengths and weaknesses of this approach and proposes some alternate approaches for defining athletic conditioning relevant to cardiovascular adaptation.

WHAT DETERMINES CARDIAC REMODELING?

Like most biological systems, the heart adapts to the physical and metabolic load placed upon it. During dynamic exercise, the wall stress of both ventricles increases as a result of increases in pressure and volume,[4,5] and it would seem that similar hemodynamic stressors are also incurred in the atria.[6] Echocardiography and cardiac MRI have been used to demonstrate that ventricular volumes increase with exercise intensity until a point approximating anaerobic threshold, from which point ventricular volumes reduce slightly.[4,7] In a similar but not identical manner, ventricular afterload increases with exercise intensity in a near-linear manner such that the load against which the two ventricles eject increases progressively with exercise until the point of fatigue.[5,8] In combination, both a volume and pressure load are imposed on the myocardium that increases such that the greatest load, and presumably the greatest stimulus for structural and functional adaptation, occurs during moderate-to-high intensity dynamic exercise (Fig. 1). A recent prospective 1-year training study supports this premise.[9]

It stands to reason that a second major determinant of the extent of cardiac remodeling would be the time that the myocardium is exposed to increased hemodynamic stress. This concept may be inferred from the finding that endurance athletes have greater cardiac remodeling when compared with athletes from sprint or team sports.[2] The observed differences in cardiac size have often been attributed to unique hemodynamic conditions created from varied combinations of static and dynamic exercise components. However, it is at least as likely that the remodeling is greater in endurance athletes simply because the hemodynamic stress is incurred for longer. For example, a typical training regime for a soccer player may involve a few hours of training per day of which the athlete may be exercising at moderate or strenuous intensity for around 60 to 90 minutes. In contrast, an endurance cyclist, rower or cross-country skier typically trains at moderate-to-high intensity for several hours each day. Thus, the cumulative time in which the heart is exposed to high hemodynamic stress is likely to be many-fold higher (Fig. 2). This fact, although seldom discussed in the literature, is likely to be

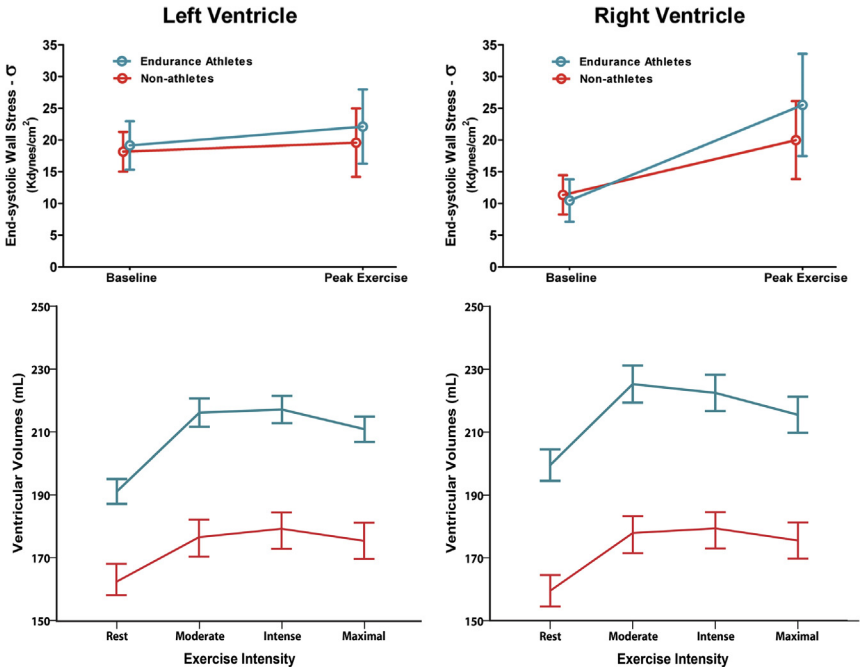

Fig. 1. A combination of invasive pressure measures and cardiac volumes obtained during exercise enable one to understand the hemodynamic stressors on the left and right ventricles. In both chambers, both the pressure and volume loads increase during exercise, and this increase is greatest at moderate to strenuous exercise intensities.