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Athlete's Heart: Is the Morganroth Hypothesis Obsolete?

Q1 **Mark J. Haykowsky [a,b*], T. Jake Samuel [c], Michael D. Nelson [c], Andre La Gerche [b,d]**

Q2 [a]Integrated Cardiovascular Exercise Physiology and Rehabilitation (iCARE) Laboratory, College of Nursing & Health Innovation, University of Texas Arlington, Arlington, TX, USA
Q3 [b]Sport Cardiology, Baker Heart and Diabetes Research Institute, Melbourne, Vic, Australia
Q4 [c]Applied Physiology and Advanced Imaging Laboratory, College of Nursing & Health Innovation, University of Texas Arlington, Arlington, TX, USA
[d]Department of Cardiovascular Medicine, University of Leuven, Belgium

In 1975, Morganroth and colleagues reported that the increased left ventricular (LV) mass in highly trained endurance athletes versus nonathletes was primarily due to increased end-diastolic volume while the increased LV mass in resistance trained athletes was solely due to an increased LV wall thickness. Based on the divergent remodelling patterns observed, Morganroth and colleagues hypothesised that the increased ''volume'' load during endurance exercise may be similar to that which occurs in patients with mitral or aortic regurgitation while the ''pressure'' load associated with performing a Valsalva manoeuvre (VM) during resistance exercise may mimic the stress imposed on the heart by systemic hypertension or aortic stenosis. Despite widespread acceptance of the four-decade old Morganroth hypothesis in sports cardiology, some investigators have questioned whether such a divergent ''athlete's heart'' phenotype exists. Given this uncertainty, the purpose of this brief review is to re-evaluate the Morganroth hypothesis regarding: i) the acute effects of resistance exercise performed with a brief VM on LV wall stress, and the patterns of LV remodelling in resistance-trained athletes; ii) the acute effects of endurance exercise on biventricular wall stress, and the time course and pattern of LV and right ventricular (RV) remodelling with endurance training; and iii) the value of comparing ''loading'' conditions between athletes and patients with cardiac pathology.

Keywords Morganroth hypothesis • Athlete's heart • Wall stress

Introduction

Q6 The seminal 'athletes heart' study published over four decades ago by Morganroth et al. demonstrated that, compared to age and sex-matched nonathletic controls, endurance trained athletes had increased left ventricular (LV) mass that was primarily due to an increased LV end-diastolic volume [1,2]. In contrast, the increased LV mass in resistance trained athletes versus age-matched nonathletic controls was solely due to increased LV septal and posterior wall thickness [1,2]. Based on the dichotomous LV remodelling patterns, Morganroth and colleagues hypothesised that the endurance training-mediated haemodynamic (volume) load is similar to that found in patients with aortic or mitral regurgitation [1,2]. Moreover, the resistance training-mediated haemodynamic (pressure) load associated with performing a strenuous Valsalva manoeuver (VM) was proposed to be similar to that found in systemic hypertension or in patients with aortic stenosis [1,2].

Q7 Despite widespread acceptance of the Morganroth hypothesis [3], some investigators have questioned whether resistance exercise performed with a brief VM is exclusively a ''pressure overload'' stress [4–8], or that endurance exercise is primarily a ''volume overload'' stimulus [9]. Given this uncertainty, the purpose of this brief review is to re-evaluate the Morganroth hypothesis regarding: i) the acute effects of resistance exercise performed with a brief VM on LV wall stress, and the patterns of LV remodelling in resistance-trained athletes; ii) the acute

Q5 *Corresponding author at: College of Nursing & Health Innovation, University of Texas Arlington, Box 19407, 411 South Nedderman Drive, Arlington, TX, 76019, USA. Tel.: +1 817 272 5541, Fax: +1 817 272 5006. Email: mark.haykowsky@uta.edu

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effects of endurance exercise on biventricular wall stress, and the time course and pattern of LV and right ventricular (RV) remodelling with endurance training; and iii) the value of comparing "loading" conditions between athletes and patients with cardiac pathology.

Acute Effects of Resistance Exercise on Ventricular Wall Stress

In accordance with Laplace's law, LV meridional wall stress is often simplified as a function of systolic arterial blood pressure and LV geometry. More correctly, LV wall stress should consider the forces on both sides of the LV wall. Specifically, LV wall stress is a function of the difference between intracavity pressure and intrathoracic pressure (e.g. transmural pressure [10,11]) and LV geometry. The Morganroth hypothesis failed to consider changes in intra-thoracic pressure during resistance exercise as an influence on the haemodynamic forces which determine LV remodelling.

During resistance exercise performed with a brief VM — an obligatory response during repetitive sub-maximal exercise performed to failure, or when lifting a weight ~85% maximal voluntary contraction [12] — large increases in intrathoracic pressure also affect transmural pressure [5,13]. Indeed, Lentini et al. reported that the transient and marked increase in systolic blood pressure during dynamic sub-maximal (95% one-repetition maximum, 1RM) bilateral leg-press exercise performed to volitional exhaustion (rest: 160 mmHg vs. exercise: 270 mmHg) was primarily due to the marked increase in intrathoracic pressure associated with performing a forceful VM (rest: 0.8 mmHg vs. exercise: 58 mmHg) [13]. Notably, LV systolic transmural pressure during leg press exercise performed to volitional exhaustion was 24% lower than that predicted by the systolic blood pressure alone [13].

Currently, only one study has measured LV wall stress during resistance exercise performed with a brief VM [5]. Haykowsky et al., using transthoracic echocardiography combined with invasive haemodynamic and intra-thoracic pressure monitoring, reported that submaximal (80% 1RM: 338 kg × 9 repetitions, and 95% 1RM: 401 kg × 4 repetitions) and maximal (420 kg × 1 repetition) bilateral leg-press exercise performed with a brief VM was not associated with an increase in LV end-systolic wall stress compared to rest in younger healthy males (Figure 1) [5]. This finding may explain why concentric hypertrophy is not an obligatory adaptation in resistance trained athletes [6,13]. It also challenges the orthodox management of patients with some valvular pathologies and aortic disease in which it is recommended that strength and power training should be avoided. Given that increases in intrathoracic pressures tend to attenuate transmural pressure, the effective stress on these pathologies would be expected to be modest. Although it would perhaps be most prudent to validate the findings of

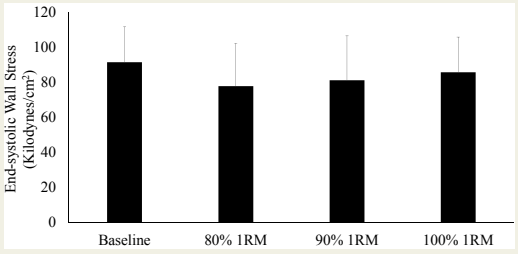

Figure 1 Left ventricular end-systolic wall stress during leg press exercise performed with a brief Valsalva manoeuvre.
Data are mean ± SD; Adapted from published data by Haykowsky et al. [5].

Hakowsky et al. [5] prior to changing current recommendations, it seems that there is good reason to challenge the current mantra.

In summary, acute heart-lung interactions are often not accounted for but remain an important determinant of LV wall stress during resistance exercise performed with a brief VM, and erroneous conclusions with respect to LV wall stress quantification can occur when positive swings in intrathoracic pressure and transmural pressure are not accounted for.

Patterns of Ventricular Remodelling With Resistance Training

Several cross-sectional or longitudinal echocardiographic studies have demonstrated that resistance training is not associated with a change in LV wall thickness, cavity size, or mass in healthy young, middle-aged or older men or women [14–18]. In contrast, a meta-analysis by Utomi et al. found that the increased LV mass in male endurance trained (n = 64 studies, 1099 participants) or resistance trained (n = 25 studies, 510 participants) athletes compared with sedentary controls (n = 59 studies, 1239 participants) was due to increased LV diastolic cavity dimension, posterior wall thickness and ventricular septal wall thickness [19]. Notably, the pattern of LV remodelling observed in resistance trained athletes was eccentric hypertrophy, not dissimilar, although smaller in magnitude, to that found in endurance athletes [19].

Given that resistance trained athletes from diverse sporting disciplines (e.g., bodybuilding, weightlifting, powerlifting) vary with respect to the type of strength exercises performed, absolute amount of weight lifted, number of sets and repetitions and rest between lifts, training sessions per week and caloric intake, it is likely that the pattern of LV remodelling between these athletes may not be homogeneous. Indeed, in a systematic review assessing the patterns of LV remodelling in resistance trained athletes, the most common patterns were normal geometry (37.5% of studies, most common in powerlifters) and concentric LV hypertrophy (37.5% of studies, most

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