

Athlete's Heart: Is the Morganroth Hypothesis Obsolete?



Mark J. Haykowsky, PhD^{a,b*}, T. Jake Samuel, MSc^c,
Michael D. Nelson, PhD^c, André La Gerche, MBBS, PhD^{b,d}

^aIntegrated Cardiovascular Exercise Physiology and Rehabilitation (iCARE) Laboratory, College of Nursing & Health Innovation, University of Texas Arlington, Arlington, TX, USA

^bSport Cardiology, Baker Heart and Diabetes Research Institute, Melbourne, Vic, Australia

^cApplied Physiology and Advanced Imaging Laboratory, College of Nursing & Health Innovation, University of Texas Arlington, Arlington, TX, USA

^dDepartment 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

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 manoeuvre (VM) was proposed to be similar to that found in systemic hypertension or in patients with aortic stenosis [1,2].

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

*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

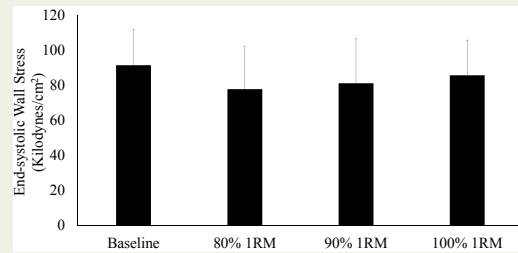


Figure 1 Left ventricular end-systolic wall stress during leg press exercise performed with a brief Valsalva manoeuvre.

Data are mean ± SD; Adapted from published data by Haykowsky et al. [5].

Haykowsky 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

common in weightlifters) with 25% of athletes displaying eccentric LV hypertrophy (most common in bodybuilders) [6]. The heterogeneous LV remodelling may be the result of the diverse cardiac load imposed on the heart during resistance training. Specifically, peak stroke volume and cardiac output are greater in bodybuilders compared to powerlifters during unilateral knee extension and squat exercise (50%, 80%, 100% one repetition maximum) [20], a finding that may explain why eccentric hypertrophy is common in bodybuilders [6]. A second explanation for the divergent patterns may be related to the use of anabolic steroids which is associated with concentric hypertrophy [21]. Finally, some resistance trained athletes may be predisposed to having larger LV wall thickness and mass that is magnified with intense training [6].

Acute Effect of Endurance Exercise on Ventricular Wall Stress

Morganroth et al. suggested that endurance exercise is primarily a “volume” load stress, however Stickland et al, using invasive haemodynamic monitoring at rest and during upright cycle exercise in younger LO ($n = 3$, VO_{2max} : 43 ml/kg/min) and HI fit ($n = 5$, VO_{2max} : 60 ml/kg/min) male subjects, found that mean pulmonary artery pressure increased by 61% and 129% from rest to submaximal and peak exercise, respectively while end-systolic pressure increased by 32% and 42% [22]. A consequence of the heightened biventricular “pressure” load is that it may result in a concomitant increase in RV and LV end-systolic wall stress.

La Gerche et al. measured RV and LV end-systolic wall stress at rest and during maximal semi-supine cycle exercise in endurance trained athletes (VO_{2max} : 58 ml/kg/min) and age-sex-matched non-athletes (VO_{2max} : 34 ml/kg/min) [23]. In all subjects, resting end-systolic wall stress was 43% lower in the RV compared to the LV, and increased to a greater extent in the RV (125%) compared to the LV (14%) during

peak exercise as a result of a greater increase in pulmonary artery systolic pressure compared to systolic blood pressure (166% vs. 36%, respectively, Figure 2) [23]. Although endurance trained athletes had a larger relative increase in pulmonary artery systolic pressure and RV end-systolic wall stress versus non-athletes, no significant difference was found between groups at the same power output [23]. Thus, contrary to the Morganroth hypothesis, endurance exercise is associated with both a ‘pressure’ and ‘volume’ load stress. Moreover, the magnitude of the increase in RV end-systolic wall stress appears dependent on exercise intensity [23].

Time Course and Pattern of Ventricular Remodelling With Endurance Training

Several recent studies have examined the time course and pattern of ventricular remodelling associated with endurance training [9,24]. Arbab-Zadeh et al. assessed RV and LV adaptations in response to a 12-month progressive endurance exercise training in 12 (mean age: 29 years, seven men and five women) previously sedentary subjects [9]. The main finding was that during the first 6 months, when lower intensity exercise was performed, the increased LV mass was solely the result of an increase in LV mean wall thickness (concentric hypertrophy) [9]. Thereafter, when high-intensity interval and longer duration endurance exercise was performed, the LV dilated and returned the mass to volume ratio close to baseline (eccentric hypertrophy) [9]. In contrast, RV volume and mass increased throughout the training program resulting in no significant change in mass to volume ratio (eccentric hypertrophy) [9]. As highlighted above, this finding is likely due to relatively greater end-systolic wall stress imposed on the RV compared to the LV during endurance exercise [23].

More recently, Weiner et al. examined the LV remodelling pattern in response to a short-term endurance training (acute augmentation phase, 12.6 hours/week and supplemental

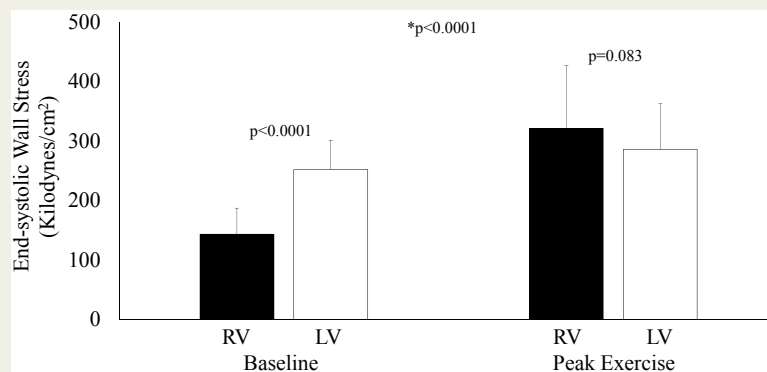


Figure 2 Changes in right and left ventricular end-systolic wall stress during maximal cycle exercise. Data are mean \pm SD; *, p value for interaction between exercise (within-subject factor) and sub-group (right ventricle vs. left ventricle as between-subject factors) on mixed-factorial analysis of variance; Adapted from published data by La Gerche et al. [23].

Table 1 The Morganroth hypothesis revisited.

Morganroth Hypothesis (Old)	Revised Physiology (New)	Clinical Relevance
Resistance and endurance training cause divergent patterns of remodelling.	Athletic cardiac remodelling is defined by the amount of haemodynamic stress exposure (time x intensity of sport) and correlates with fitness.	When assessing a patient with cardiac hypertrophy, the clinician should not be expecting significant concentric remodelling in athletes, resistance or endurance trained, and should consider pathological causes.
Resistance training with a VM causes a similar haemodynamic stress to hypertension or aortic stenosis.	Resistance training with a brief VM causes a transient increase in intravascular pressure. Transmural pressure modestly increased due to counteracting increases in intrathoracic pressure.	Resistance training with a brief VM would be expected to have modest impact in patients with aortic disease (e.g. Marfan syndrome or bicuspid aortopathy). Although a cautious approach is prudent, a reappraisal of orthodox advice is warranted.
Did not consider the RV	The RV seems to be exposed to a disproportionate increase in wall stress during exercise resulting in slightly greater chronic remodelling.	RV dilation is common in endurance athletes and often is more prominent than the extent of LV dilation. Mildly asymmetric remodelling should be considered normal in athletes.
Did not consider stages of training	Both LV and RV remodelling change with time, training load and level of previous training.	Cardiac remodelling in young athletes may differ from veteran athletes in both the pattern and extent of hypertrophy.

Abbreviations: RV, right ventricular; LV, left ventricular; VM, Valsalva manoeuvre.

strength training 1 hours/week for 90 days) followed by a chronic maintenance phase (12.2 hours/week and supplemental strength training 1.1 hours/week for 36 months) among 12 competitive male rowers (mean age: 19 years) [24]. Compared to baseline, the increased LV mass during the acute augmentation phase was secondary to an increase in LV end-diastolic volume with no change in LV wall thickness [24]. In turn, the continued increase in LV mass during the chronic maintenance phase was the result of an increase in LV wall thickness [24]. The disparity between these findings and those of Arbab-Zadeh et al. were attributed to the underlying training status (endurance trained athletes with substantial baseline exercise exposure of ~8 hours/week vs. previously sedentary subjects) and training stimulus (relatively fixed high-intensity/high-volume versus a progressive incremental load protocol). Despite these differences, and contrary to the Morganroth hypothesis, both studies demonstrate that endurance training is associated with a biphasic LV remodelling pattern that appears to be related to the underlying training load (e.g. greater pressure load associated with lower intensity sub-maximal exercise and greater volume load coupled with a continued pressure load associated with high-intensity exercise [4]) and prior exercise training exposure.

Ventricular Wall Stress in Athletes Versus Patients With Cardiac Pathology: Intermittent Versus Permanent Load

A key factor unique to athletic remodelling but not considered in the Morganroth hypothesis is that the haemodynamic load

imposed by exercise is a transient phenomenon. Specifically, given that the haemodynamic stimulus for ventricular remodelling is only present during exercise, the extent of remodelling is probably influenced by the percentage of time spent exercising. This may well explain why those athletes who spend the greatest amount of time training and racing (e.g. cyclists, cross-country skiers, and triathletes) tend to have the most profound exercise-induced remodelling [25]. This important dimension of “time” needs to be considered when drawing an analogy between cardiovascular pathologies such as hypertension and valvular heart disease in which the load is “constant” and the athlete’s heart where the load is “intermittent”.

Conclusion

Despite being widely accepted in the sport cardiology field [3], we contend that the four-decade-old Morganroth hypothesis that resistance exercise performed with a brief VM is primarily a ‘pressure’ load stress (similar to aortic stenosis or long standing hypertension) that results in concentric LV hypertrophy is obsolete (Table 1) [5–8]. Further, contrary to the Morganroth hypothesis, endurance exercise is associated with both an acute “volume” and “pressure” load [4,22,23]. Importantly, the finding that end-systolic wall stress is greater for the RV compared to the LV secondary to a greater relative increase in pulmonary artery systolic pressure compared to systolic arterial blood pressure, suggests that the RV is also subject to a “pressure” load during exercise. Finally, the time course and pattern of ventricular remodelling appears to be related to underlying training load (intensity and duration) and prior training exposure [9,24].

In closing, the Morganroth hypothesis was derived from hypotheses rather than direct measures of ventricular wall stress during resistance or endurance exercise. The important dimension of "time" should be considered when drawing an analogy between cardiovascular pathology (e.g. hypertension, valvular disease) where the load is *constant* and the athlete's heart where the load is *intermittent*. Future studies examining the pattern of ventricular remodelling need to account for the acute and chronic effects of the sport (exercise) in question in relation to RV and LV wall stress and its determinants (transmural pressure, ventricular geometry). Failure to do so may result in erroneous conclusions.

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