



## Athlete's heart and the Morganroth hypothesis



The four-decade-old "Morganroth hypothesis" that resistance exercise performed with a brief Valsalva manoeuvre (VM) is primarily a "pressure" load stress – similar to aortic stenosis or long standing hypertension – that results in concentric left ventricular (LV) hypertrophy is obsolete," according to a review paper to appear in the journal *Heart, Lung and Circulation*.

"Further, contrary to the Morganroth hypothesis, endurance exercise is associated with both an acute 'volume' and 'pressure' load," write Mark J. Haykowsky, PhD, MSc, Integrated Cardiovascular Exercise Physiology and Rehabilitation (iCARE) Laboratory, College of Nursing & Health Innovation, University of Texas Arlington, and co-authors.

The seminal "athlete's heart" study (1975), by Morganroth and colleagues, reported that the increased 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 et al. 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 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. In this review, Haykowsky and co-authors sought to re-evaluate the Morganroth hypothesis regarding:

1. 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;
2. 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
3. The value of comparing "loading" conditions between athletes and patients with cardiac pathology.

According to the paper, the Morganroth hypothesis failed to consider changes in intrathoracic pressure during resistance exercise as an influence on the haemodynamic forces which determine LV remodelling. The article explains that "erroneous conclusions with respect to LV wall stress quantification can occur when positive swings in intrathoracic pressure and transmural pressure are not accounted for."

Meanwhile, 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, the article points out.

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, the paper notes. In addition, the time course and pattern of ventricular remodelling appears to be related to underlying training load (intensity and duration) and prior training exposure.

The paper also explains that "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."

Finally, the paper says 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," the paper adds.

Source: [Heart, Lung and Circulation](#)

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