PERSPECTIVE

Left ventricular remodelling and the athlete's heart: time to revisit the Morganroth hypothesis

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A widely held belief in sport cardiology is that the pattern of left ventricular (LV) remodelling associated with athletic training depends on the type of physical conditioning performed. This is based on the 1975 echocardiographic study by Morganroth *et al.* who found that male collegiate endurance trained (runners and swimmers) athletes had increased LV diastolic cavity size and mass with a normal wall thickness (eccentric hypertrophy) compared to age- and sex-matched non-athletic control subjects (Morganroth *et al.* 1975). In contrast, resistance trained athletes (wrestlers and shot putters) had increased LV wall thickness and mass with normal diastolic cavity size (concentric hypertrophy) compared to controls (Morganroth *et al.* 1975). The stimulus for physiological eccentric hypertrophy was attributed to the large LV volume loads (diastolic wall stress) present during endurance exercise while the stimulus for concentric hypertrophy was the result of intermittent increases in arterial pressure loading (systolic wall stress) associated with performing a Valsalva manoeuvre during lifting (Morganroth *et al.* 1975; Morganroth & Maron, 1977). Although evidence supports the acceptance of the 'Morganroth hypothesis' for endurance training, uncertainty remains if LV remodelling is an obligatory adaptation with resistance training (Haykowsky *et al.* 2002; Naylor *et al.* 2008).

In a recent issue of *The Journal of Physiology*, Spence *et al.* (2011) report new data that challenge, at least for resistance training, the Morganroth hypothesis. The authors examined the effects of 6 months of endurance training (3 hours per week of walking/jogging/running, $n = 10$) or resistance training (3 hours per week of Olympic weightlifting and supplemental strength exercises, $n = 13$) followed by 6 weeks of detraining on LV morphology and function, aerobic fitness, muscle strength and body composition in younger males (mean age: 27 years). Cardiac magnetic resonance imaging (MRI) and speckle tracing echocardiography were used to assess LV morphology and function. The paper provides novel information because it is the first to directly compare the two forms of exercise using MRI, which is a much more accurate assessment of cardiac morphology than previously utilized methods (Bellenger, *et al.* 2000) and because it adopted a within subjects longitudinal approach, which is less affected by the limitations of scaling than previous athletic comparisons.

Spence *et al.* reported that total maximal strength and lean mass were significantly higher after resistance training and detraining compared to baseline with no change in LV morphology and function, or aerobic fitness. In contrast, cardiac MRI derived LV mass and aerobic fitness increased significantly after endurance training and returned towards baseline after detraining. Endurance training also significantly increased LV septal wall thickness and end-diastolic volume ($P = 0.05$) while posterior wall thickness decreased significantly after detraining. Finally, total lean mass and strength were higher after endurance training with the former returning to baseline after detraining.

The LV remodelling pattern observed by Spence *et al.* after 6 months of endurance training is consistent with that expected based on the Morganroth hypothesis. However, their finding that resistance training did not significantly change LV posterior or interventricular septal wall thickness or mass contradicts the expected effect for this type of exercise based on Morganroth's hypothesis. A reason for this finding is that resistance training may not acutely increase in LV systolic wall stress. Indeed, our research group previously reported that (sub)maximal leg-press exercise performed with a 2–3 s Valsalva manoeuvre was not associated with an alteration in LV end-systolic wall stress in younger healthy males (mean age: 27 years) (Haykowsky *et al.* 2001). This observation combined with the new cardiac MRI findings by Spence *et al.* suggests that the four-decade old Morganroth hypothesis with respect to resistance training mediated-LV remodelling needs revisiting.

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