Exercise and cardiac adaptations

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In the 1970s, Morganroth *et al.* (1975) proposed two distinct forms of hypertrophy related to remodeling of athletes hearts; concentric or eccentric hypertrophy. These adaptations can be rationalized by the Law of La Place which implies that to maintain a constant relationship between chamber pressure and the ratio of wall thickness to ventricular radius, cardiac muscle grows to match the workload imposed on the ventricle. This proposal has become accepted textbook dogma. However due to different techniques and use of different (usually rather small) study populations, the veracity of the Morganroth proposal needs to be revisited, especially in as it pertains to LV remodelling in response to resistance training. We present new MRI based data pertaining to the effects of intense exercise of different modalities on cardiac morphological adaptation which indicates that intense endurance exercise resulted in a significant increase in LV mass (112.5±7.3 to 121.8±6.6g, $P<0.01$) which reversed following a period of detraining. Interestingly, resistance training did not impact the mass of the LV (125.7 ± 7.6 to 130.3 ± 6.4g) (Spence *et al.*, 2011). These data cast further doubt over the proposal that remodelling occurs in response to resistance training.

Concomitant with the morphological adaptations, changes in cardiac function, particularly diastolic function, have also been observed in athletes. Since acute bouts of exercise are associated with enhanced diastolic filling, repetitive bouts most likely cause physiological adaptations which enhance indices of diastolic function. However, cross-sectional studies suggest that the indices of diastolic function remain unchanged, or only modestly increased in athletes compared with controls. With the recent availability of more accurate technological approaches, such as myocardial tissue and speckle echocardiography, which are independent of loading conditions, a better understanding of exercise induced physiological adaptations on function is emerging. Although rare, longitudinal studies performed in athletes, provide insight into changes in diastolic function in athletes. In a study conducted by our group, athletes were assessed across an intensive training cycle following a period of relative detraining (Naylor *et al.*, 2005). Diastolic function was impaired after a period of detraining in these athletes who exhibited LV hypertrophy. Resumption of training further increased LV mass, which was associated with improved diastolic indices. These studies raise the intriguing hypothesis that diastolic function may be normal in the athletes so long as the training stimulus is extant.

Given that exercise training is imperative in maintaining optimal diastolic function in athletes who display cardiac hypertrophy, it stands to reason that exercise training may be able to augment diastolic function in other population groups with cardiac hypertrophy. Exercise has been documented to produce improvements in cardiac function in adults with previously impaired diastolic function (Forman *et al.*, 1992; Levy *et al.*, 1993). Further support for the use of exercise interventions to improve diastolic function comes from evidence that shows a direct relationship between early diastolic tissue velocity (E') and exercise capacity (Wong *et al.*, 2005; Grewal *et al.*, 2009). This improvement is primarily due to peripheral adaptations, but a large component can also be attributed to improvements in diastolic function resulting from exercise (Belardinelli *et al.*, 1995).

Indeed, our group observed an improvement in diastolic function in obese adolescents with pre-existing cardiac hypertrophy following an exercise training program (Naylor *et al.*, 2008). Furthermore, our results suggested that the enhanced diastolic indices observed after exercise training are likely due to a change in intrinsic properties of the myocardial wall rather than simply changes in loading conditions. Similar results have been reported in other populations, such as dilated cardiomyopathy (Belardinelli et al., 1995). This can further be supported by animal studies which indicate high-intensity training can reduce ventricular stiffness *via* an intrinsic effect, independent of neurohormones, heart rate, or pericardial restraint (Libonati *et al.*, 2005).

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