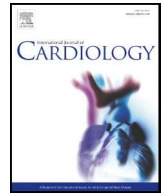




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Characterization of the dynamic changes in left ventricular morphology and function induced by exercise training and detraining

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ABSTRACT

Background: Although exercise-induced cardiac hypertrophy has been intensively investigated, its development and regression dynamics have not been comprehensively described. In the current study, we aimed to characterize the effects of regular exercise training and detraining on left ventricular (LV) morphology and function.

Methods: Rats were divided into exercised ($n = 12$) and control ($n = 12$) groups. Exercised rats swam 200 min/day for 12 weeks. After completion of the training protocol, rats remained sedentary for 8 weeks (detraining period). Echocardiographic follow-up was performed regularly to obtain LV long- and short-axis recordings for speckle-tracking echocardiography analysis. Global longitudinal and circumferential strain and systolic strain rate were measured. LV pressure-volume analysis was performed using additional groups of rats to obtain haemodynamic data.

Results: Echocardiographic examinations showed the development of LV hypertrophy in the exercised group. These differences disappeared during the detraining period. Strain and strain rate values were all increased after the training period, whereas supernormal values rapidly reversed to the control level after training cessation. Load-independent haemodynamic indices, e.g., preload recruitable stroke work, confirmed the exercise-induced systolic improvement and complete regression after detraining.

Conclusions and translational aspect: Our results provide the first comprehensive data to describe the development and regression dynamics of morphological and functional aspects of physiological hypertrophy in detail. Speckle-tracking echocardiography has been proven to be feasible to follow-up changes induced by exercise training and detraining and might provide an early possibility to differentiate between physiological and pathological conditions.

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1. Introduction

Long-term, regular physical training results in structural and functional alterations in the heart that are adaptive responses to the increased haemodynamic demands during exercise sessions. This complex adaptive cardiac remodelling has been referred to as “athlete's heart” and includes a balanced enlargement of ventricular cavity dimensions and myocardial mass, accompanied by increased stroke volume and lower resting heart rate [1,2]. The recognition of training-induced physiological hypertrophy has attracted numerous experimental and clinical projects.

These left ventricular (LV) structural adaptations in response to exercise training and alterations after cessation of training (detraining) have been confirmed utilizing echocardiography several decades ago [3]. Since then, several experimental and human studies reflected that after inducing myocardial hypertrophy by exercise training, short- and long-term deconditioning results in progressive regression of exercise-induced cardiac morphological remodelling [4–6]. This phenomenon might be especially valuable to distinguish between athlete's heart and structurally similar forms of pathological hypertrophy in case of challenging clinical cases, the so-called grey-zone hypertrophy [7].

Currently, the impact of exercise training on LV myocardial mechanics is less clear. In the 20th century, the echocardiographic studies suggested unchanged resting LV systolic function using traditional parameters, which was confirmed by cardiac magnetic resonance imaging [8,9]. These non-invasive techniques have an essential role in describing the morphological characteristics of the athlete's heart; however, they are not able to characterize cardiac intrinsic LV systolic

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