THE CHANGES OF SERUM TESTOSTERONE IN INJURY OF HEART OF EXHAUSTIVE EXERCISED RATS

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Objectives To study the changes of serum testosterone in heart injury of exhaustive rats, therefore provide information for
prevention and treatment of heart injury.

Methods 20 male Sprague-Dawley rats were randomly divided into quiet control (C) group (n=10), one-time exhaustive exercise (E) group (n=10). Rats were killed after single bout of exhaustive swimming. Group C and Group E were gavaged with distilled water for 10 days. Group C weren’t treated. Group E underwent one-time exhaustive swimming. Each group was narcotised with 10% hydral and serum specimen was taken to measure the activity of MB isoenzyme of creatine kinase (CK-MB) and levels of cardiac troponin I (cTnI) and testosterone. Myocardium samples were taken to observe histomorphologic changes by light microscope and electron microscope afterwards.

Results
1. Serum cTnI assay results: The level of serum cTnI of Group E is significantly higher than Group C (1.4077±1.3086: 0.5141 ±0.3677 ng/ml, p<0.05).
2. Serum CK-MB assay results: Serum CK-MB activity of Group C is significantly lower than Group E (568.70±107.09: 1547.80 ±393.62 U/L, p<0.01).
3. Serum testosterone assay results: Serum testosterone level of Group C is significantly higher than Group E (2.73±0.51:0.82 ±0.11, 1.24±0.12 ng/ml, p<0.01).
4. Histomorphology of myocardium samples observed by light and electron microscopy: cardiac sarcomeres of Group C rat were arranged in neat rows, the density was uniform and there was no organelle oedema, mitochondrial membrane and ridge were normal; The matrix of myocardium of Group E suffered a high degree of oedema and wider perinuclear gap. The number of mitochondria and glycogen reduced significantly. Part of the ridge and part of membrane mitochondria fused, blurred or missed, a small amount of myocardium underwent fibre necrosis.

Conclusions Exhaustive exercise has a significant injury to rat myocardium. These effects possible work through decrease serum testosterone therefore injuring heart. Testosterone has important effect on human body metabolism, physiology and pathology of the heart. Higher concentrations of testosterone or its chronic effects can increase T-, L-type calcium channels density, and low concentrations or acute effects can block T-, L-type calcium channel, reduce the male Q-Tc period, and improve the sensitivity to insulin and lipid metabolism. Testosterone can increase the calcium regulatory proteins and expression of β-2 receptor. It can increase the rate of calcium transient and reduce the calcium over-load in the case of increasing the intracellular calcium concentration. A certain concentration of testosterone can sustain a certain vascular tension, improve the cardiac conduction or dilate coronary, reduce insulin resistance and incidence of metabolic syndrome, improve the myocardial ischaemia, reduce apoptosis and fibrosis of myocardial cell, protect the heart and improve the efficiency of cardiac diastolic. This research may provide reference for prevention and treatment of myocardial injury induced by exhaustive exercise in athletes and military men.