Distance running training causes cardiac hypertrophy which is not associated with impaired left ventricular (LV) function. Debates continue whether regular strength training triggers more concentric cardiac hypertrophy than aerobic training, and how it affects LV function. Strongman sport is a violent physical activity during which haemodynamic pressure overload predominates. The aim of our study was to compare LV structure and function, as well as cardiovascular risk profile, between strongmen, long-distance runners, and sedentary controls.

Methods. Cardiovascular ultrasonography and venous blood analysis were performed in nine strongmen and 10 marathon runners of similar sporting ambitions, career experience and training devotion, as well as 10 sedentary controls. All subjects were adult males, and groups were age-matched. Controls were heavier than runners but lighter than strongmen (p<0.001). Maximal oxygen uptake was about twice higher in runners than in either other group.

Results. Absolute LV diameter in strongmen was similar (p>0.05) to that of runners, but higher (p<0.05) than in controls. Relative LV diameter was higher (p<0.01) in runners as compared with strongmen or controls. Absolute LV wall thickness was higher (p<0.01) in athletes than controls, while relative LV wall thickness was higher (p<0.001) in runners as compared with either other group. Absolute LV mass was higher (p<0.05) in strongmen and runners as compared with controls. However, LV mass index was greater (p<0.001) in marathoners as compared with strongmen and controls. Relative wall thickness (the sum of interventricular septum and LV posterior wall thickness divided by LV diameter) did not differ between the groups. Body surface area-indexed diameters of atriums, right ventricle, aorta, and pulmonary artery were increased (p<0.01) in runners as compared with either other group. Ejection fraction was higher (p<0.05) in runners than other groups. Diastolic LV function was deteriorated in strongmen: their Doppler E-peak was lower than in runners (p<0.001) or controls (p=0.002), and A-peak was higher than in runners (p=0.002) or controls (p<0.05). Runners had higher E/A ratio than strongmen (p<0.001) or controls (p<0.05). Flow-mediated dilation of the brachial artery and blood triglyceride level did not differ between the three groups. Low-density-lipoprotein cholesterol was higher (p<0.05) in strongmen as compared to runners, while marathoners had higher (p<0.001) high-density-lipoprotein cholesterol than strongmen but not controls. The coefficient of atherogenicity was lower (p<0.05) in marathoners vs. controls, and in controls vs. strongmen.

Conclusions. Strongmen and marathon runners have similar absolute cardiac dimensions and structural type, but marathoners possess far greater heart size in relative terms. Diastolic LV function is deteriorated in strongmen, but not in marathoners. Strongmen possess less favourable blood lipid profile as compared with runners.

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