

Case report

Cardiac parameters and endothelial function in a strength athlete: a case report

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Abstract — Aims: We aimed to discuss a case of strength training athlete who competes in international competitions regarding cardiac (dimension and function), vascular (endothelium and vascular resistance), hemodynamic (blood pressure), given limited evidence supporting these cardiovascular adaptations as well as concerning endothelial function in long-term high-intensity strength training. **Methods:** We assessed heart structure and function (echocardiography); systolic (SBP) and diastolic blood pressure (DBP); endothelium-dependent vasodilation (flow-mediated dilation, FMD); maximum force tested in the squat, bench press, and deadlift; and maximum oxygen consumption (spirometry). **Results:** powerlifter's cardiac dimensions (interventricular septum 13 mm; posterior wall thickness 12 mm; LV diastolic diameter 57 mm; left ventricle mass 383 g; LV mass adjusted by body surface area 151.4 g/m²) are above the proposed cutoff values beyond which pathology may be considered. Moreover, cardiovascular function systolic (ejection fraction by Simpson's rule, 71%) is preserved and FMD measure is fairly close and above normal; however, a mild increase in systolic and diastolic blood pressure was observed (130/89 mmHg, respectively). **Conclusion:** Cardiac remodeling cannot be viewed as either pathological or harmful to the cardiovascular system. Furthermore, we showed an improvement in endothelial function.

Keywords: athlete's heart, ventricular hypertrophy, flow-mediated dilation, strength training.

Introduction

The heart develops anatomical and physiological adaptive changes in response to exercise training, particularly long-term aerobic exercise, that constitute a clinical entity classically called "athlete's heart"¹. Another question regarding cardiovascular changes in response to exercise training is about the existence of an "athlete artery". Studies have shown that peripheral and coronary arteries of athletes are larger than those of control subjects, and peak blood flow to the legs is enhanced in athletes suggests that resistance arteries undergo increases in their cross-sectional area. Such cardiovascular remodeling phenomenon could be a result of hemodynamic shear stress mediated by endothelial factors².

In contrast, researchers have investigated a potentially negative association between high-intensity strength training and cardiovascular health³, since it involves a great amount of slow-speed contractions using high loads close to the maximum, increasing peripheral vascular resistance (PVR), left ventricle (LV) pressure overload. Cardiovascular remodeling in response to high-intensity strength training could induce changes over time (concentric LV hypertrophy) that are similar to those seen in serious heart conditions such as hypertrophic cardiomyopathy⁴ or hypertensive disease. However, this state has not been evidenced in strength training athletes^{5,6} and some findings may be misinterpreted and lead to an incorrect diagnosis (false positive). Thus, in current clinical practice, a special attention should be given to "a misdiagnosed condition can ensure an unnecessary cessation of an exercise program"^{7,4}. This problem goes beyond

"high-performance" and also affects amateur athletes.

Due to the complexity of the research question, mainly in the difficulty of recruiting the sample with the same characteristics including do not use anabolic steroid, common among powerlifters, we presented a case study to discuss cutoff parameters structural and functional cardiac changes, blood pressure and endothelium-dependent vasodilation in a powerlifter.

Case report

RC, 35-year-old male, height 1.84 m, and weight 135 kg. The powerlifter, who competes in international competitions (including The World's Strongest Man – US). Fifteen years of powerlifting training (5 days/week; 60 minutes/day) without aerobic exercises. No prior history of cardiovascular conditions. Denies the use of anabolic steroids or any other drugs for the last 24 months. We performed a strength assessment for the following exercises: squat 390 kg; bench press 270 kg; deadlift 415 kg, and total load 1,075 kg. We conducted an echocardiographic assessment on a different day (all images were stored and sent to a second echocardiography specialist for blind evaluation of images) – Figure 1. The cutoff values for the parameters assessed are presented by specific studies with athletes according to the Recommendations for Chamber Quantification by Lang (2005) (from the American Society in conjunction with the European Association of Echocardiography) and Brazilian Guidelines of Echocardiography Indications⁸: LV ejection fraction (LVEF) (Simpson's method) 71%; preserved diastolic function; aorta 29

mm; left atrium 38 mm; interventricular septum 13 mm [cutoff values by Whyte (2004) and Lang (2005): 12-13 mm]; posterior wall thickness 12 mm [cutoff values by Lang (2005): 11 mm]; LV diastolic diameter 57 mm [cutoff values by Lang (2005): 59 mm]; LV mass 383.0 g [cutoff values by Lang (2005): 225.0 g] and LV mass adjusted by body surface area (BSA) 151.4 g/m² [cutoff values by Lang (2005): 115 g/m²]; and interventricular septum-posterior ventricular wall thickness ratio 1.08 [cutoff value by Caselli (2008): 1.03]. We calculated the relative wall thickness (RWT) by the formula $RWT = (2 \times \text{posterior wall thickness}) / \text{LV diastolic diameter}$ and found a RWT of 0.42. Thus, the athlete showed increased LV mass index and an inaccurate diagnosis of concentric hypertrophy (Figure 2) according to the Recommendations for Chamber Quantification⁷ and Brazilian Guidelines of Echocardiography Indications⁸. RC also showed 130/89 mmHg (systolic/diastolic blood pressure; duplicate measurements) and VO₂max (by spirometry) of 25.1 mL O₂/kg.min⁻¹. Finally, vascular measurements showed flow-mediated dilation (FMD, according to the literature¹¹, non-blinded evaluation) of 9.64%, and PVR of 8.00 mmHg/cm.s⁻¹.

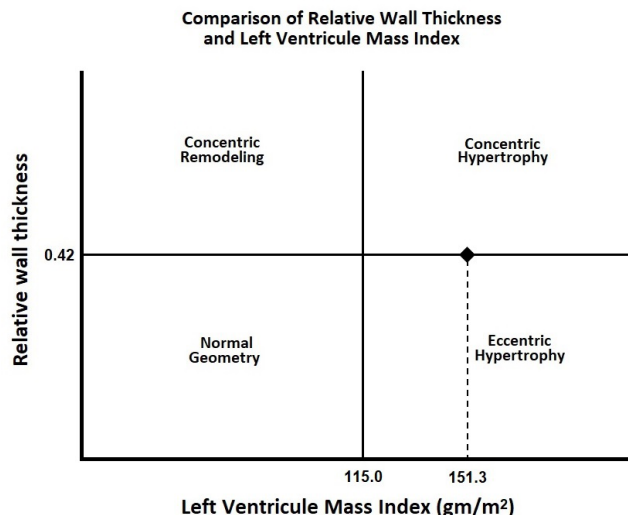


Figure 2. Relationship between relative wall thickness and left ventricle mass indexed by body surface area. The comparison permits categorization of an increase in LV mass as either concentric (relative wall thickness ≥ 0.42) or eccentric (relative wall thickness < 0.42) hypertrophy, according to Lang (2005) and Camarozano (2009). Our athlete (RC) is placed with 0.42 (relative wall thickness) and 151.5 gm/m² (left ventricle mass index).

The case report was conducted in accordance with the Ethical Principles of the Declaration of Helsinki and approved by the Research Ethics Committee at Instituto de Cardiologia do RS/ Fundação Universitária de Cardiologia (ICFUC) (protocol #417.492).

Discussion

Interestingly, the powerlifter’s cardiac dimensions are above the proposed cutoff values and could be considered a pathological state. However, RC showed that cardiovascular function is preserved. The literature has reported that both high-intensity aerobic and strength athletes exhibit increased LV parameters compared with physically inactive subjects⁷. Echocardiographic assessment based on BSA must be performed to compare individuals of different body size. A 2013 meta-analysis reported that all structural LV parameters were greater in aerobic training (AT) and strength training (ST) athletes than sedentary subjects (S) including LV mass (AT: 232, ST: 220 and S: 166 g), interventricular septum thickness (AT: 11.0, ST: 11.0 and S: 9.2 mm) and LV end-diastolic diameter (AT: 54.8, ST: 52.4 and S: 50.1 mm)¹². Based on Utomi et al.¹² proposed cutoff criteria, we found increased LV mass, intraventricular septum thickness, and LV end-diastolic diameter in the powerlifter. In contrast, LV mass, interventricular septum and LV end-diastolic diameter measures were below the upper normal cutoff values in the long-distance runner. Using a different approach, the multicenter Normal

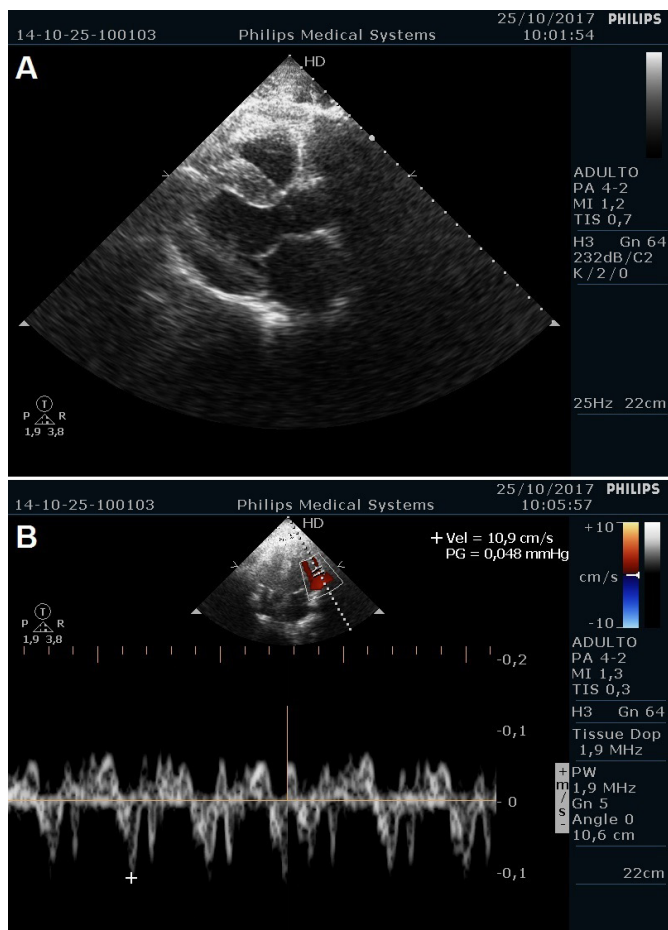


Figure 1. Echocardiographic imaging of case (powerlifter). Panel A: two-dimensional imaging using a parasternal longitudinal view. Panel B: four-chamber view on tissue Doppler for assessment of left ventricle diastolic function.

Reference Ranges for Echocardiography (NORRE) Study has proposed the following normal cutoff values for echocardiographic measurements in males: LV mass 145.6 ± 36.7 g, interventricular septum thickness 9.2 ± 1.6 mm, posterior ventricular wall thickness 9.3 ± 1.5 mm and LV end-diastolic diameter 46.2 ± 4.8 mm¹³. If we apply these criteria to our case, all cardiovascular parameters studied are increased for the powerlifter case. The finding of ventricular wall thickness in this case does not offer solid evidence to support the hypothesis of concentric hypertrophy in strength athletes¹². Other factors should be evaluated, in particular, RWT and LV mass adjusted by BSA (Figure 2)^{7,8,14}. Furthermore, preserved systolic and diastolic functions seen in the athlete, possibly, have more important implications than cardiac dimensions¹³.

Cardiac adaptive changes in long-distance runners, contradictory to the commonly accepted knowledge, seem to be biphasic: concentric hypertrophy developed first and then eccentric hypertrophy developed later¹⁵. Contradictorily, in our case study, strength training with predominantly isometric exercises would lead to increased ventricular wall thickness with no increase in chamber volume resulting from pressure overload (increased afterload) rather than volume overload due to increased blood pressure, as seen in our athlete.

Despite the above-mentioned differences in cardiac remodeling, FMD measure is fairly close and above normal¹¹ (8% for Corretti's Studies). Blood flow redistribution could in part explain similar FMD measure found in our case. It is an interesting finding given that the exercise training modality studied has quite different biomechanical when compared with aerobic exercises that knowingly improves endothelial function through increased blood flow and enhanced shear stress during exercise. A likely explanation for increased FMD values in our case is the mechanical compression of resistance blood vessels during weightlifting, followed by an abrupt release of blood flow thus producing shear stress as well².

Conclusion

Studies have shown little supportive evidence of the association of concentric hypertrophy with strength exercise, and apparently, this feature may have been misinterpreted because of assumptions based on anatomical criteria only (cardiac dimensions). Our case shows that cardiac remodeling is specific to the training modality and cannot be viewed as either pathological or harmful to the cardiovascular system. Although it is difficult to research, this subject is relevant, and it must be explored at several angles to increase the evidence of misleading clinical diagnoses.

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