

Hemodynamic and Vascular Effects of Resistance Training: Implications for Cardiovascular Disease

Daniel Umpierre e Ricardo Stein

Laboratório de Fisiopatologia do Exercício, Hospital de Clínicas de Porto Alegre - Universidade Federal do Rio Grande do Sul, Porto Alegre, RS - Brazil

Summary

Resistance training has been proposed as a possible strategy for cardiovascular prevention and rehabilitation, and in this context, this review describes the cardiovascular effects mediated by this type of intervention. Increments in both muscular strength and capacity to perform daily tasks are well-characterized benefits of this type of training. More recently, studies using hemodynamic evaluation have shown cardiovascular stability in patients with coronary disease or heart failure during the performance of resistance exercise, with no apparent detriment to ventricular function or exacerbated increase in exercise blood pressure. Additionally, resting blood pressure also seems to be influenced by chronic resistance training, with a slight reduction in both systolic blood pressure (SBP) and diastolic blood pressure (DBP). The measurement of pressure levels after a single resistance exercise session shows the occurrence of post-exercise hypotension in normal and hypertensive individuals; however, there is controversy as to the intensity of the effort necessary to induce this effect. Recently, intervention studies have investigated resistance exercise effects on vascular variables as arterial compliance and endothelial function. Despite the small number of experiments available, evidence has shown a potential influence of resistance training on the reduction of arterial compliance. On the other hand, peripheral blood flow is increased after resistance training, whereas the endothelial function seems to be improved especially after combined aerobic and resistance training. Additional research is necessary for an analysis of the efficacy of this intervention on validated outcomes, and for a greater understanding of the physiological mechanisms responsible for vascular adaptations.

Introduction

Since the mid 1960s, cardiovascular responses to predominantly strength-based exercise have been discussed^{1,2}. Until the early 1990s, resistance training (also called strength training) was not included in international guidelines. However, over the last years, this modality has come to be

Key words

Exercise/psychology; physical fitness/psychology; hemodynamic evaluation; cardiovascular diseases.

Mailing address: Ricardo Stein •

Rua João Caetano, 20/402 – Três Figueiras - 90460-270
Porto Alegre, RS - Brazil

E-mail: kuqui2@terra.com.br / rstein@cardiol.br

Manuscript received March 24, 2007; revised manuscript received May 10, 2007; accepted May 10, 2007.

considered as a possible strategy for primary and secondary prevention of different heart diseases^{3,4}. Furthermore, several research projects have suggested that resistance exercise, when appropriately prescribed and supervised, has favorable effects on different aspects of health (muscular strength, functional capacity, psychosocial well-being, besides the positive impact on cardiovascular risk factors)⁵.

Neuromuscular adaptations and effects such as an increase in muscular strength and resistance have been the primary basis for the rationale that supports the application of resistance training in exercise programs for heart patients^{4,6}. Among the different adaptations promoted for this type of training are the increased capacity to carry out daily activities⁷, an increment in tolerance to submaximal aerobic exercise^{8,9}, a suppression of age-related strength decline¹⁰, and an attenuation of cardiovascular responses to effort¹¹. Even in the absence of large clinical studies evaluating the efficacy of this type of training applied exclusively on validated clinical outcomes, observation data indicate a reduced coronary risk for resistance exercise practitioners.

In the Health Professionals Follow-up Study cohort¹², weekly participation in 30 minutes or more of resistance training was associated with a 23% decrease in risk of a non-fatal acute myocardial infarct and/or fatal cardiovascular disease (RR 0.77; 95% CI, 0.61-0.98; p=0.03). Multivariate analysis was used, adjusted for factors such as age, alcohol consumption, smoking, family history, food profile, and engagement in other physical activities to decrease the chance of systematic error.

Currently, there is further evidence as to resistance exercise in health and in cardiovascular disease (CVD) which allows a better understanding of known effects and reinforces the perspective of using resistance training in secondary prevention. In this review we discuss the influence of this type of exercise on important aspects of CVD or its development, covering evidence that is recent and/or yet unexplored in other reviews.

Hemodynamic stability during resistance exercise

According to the intensity of the effort put forth, hemodynamic responses during resistance exercise can be similar to those that occur in dynamic or isometric (static) contractions. Therefore, in efforts with low loads, there is an increase in heart rate (HR), systolic blood pressure (SBP), systolic volume, and cardiac output (CO), whereas with high loads, there is also an increase in diastolic blood pressure

(DBP)¹³. During resistance exercise, greater HR e BP values are obtained in the last repetitions of the series performed to exhaustion¹⁴, and this training design has been contraindicated for hypertensive patients. Additionally, among other factors that influence blood pressure response, the greater the muscle mass involved in the exercise, the greater the response¹⁵. MacDougall et al observed extreme pressure rises ($\approx 320/250$ mmHg) in body-builders when these individuals were exposed to loads between 80% and 100%¹⁶. On the other hand, the level of physical strength conditioning seems to maintain an inverse relationship with the magnitude of the BP and HR responses to exercise¹⁴.

In cardiac patients with left ventricular dysfunction, during strength tests and in series with different intensities, an increase in new segment abnormalities of the left ventricular wall was observed which occurred in greater proportions during the final series after a protocol of growing intensities (three series with progressive loads)¹⁷. However, these were small-magnitude findings and even if they can reflect some degree of ischemia during effort, they do not suggest a reduction in cardiac performance. Indeed, in 6,653 individuals without CVD who performed strength tests – on isokinetic dynamometer as well as in 1-maximum repetition test in exercises as bench press and leg press – there were no fatal or non-fatal cardiovascular events¹⁸.

More recently, studies with different evaluation techniques have broadened our knowledge of cardiovascular function during resistance exercise. Meyer et al¹⁹ used direct catheterization of two groups of patients: a) HF with ventricular dysfunction, and b) stable coronary artery disease with preserved ventricular function. The authors identified an increase in the cardiac index and in the systolic volume during the leg press at different effort intensities. At the 60% intensity of maximum voluntary contraction, the systolic work index remained stable, indicating similar changes in the mean blood pressure (MBP) and in the diastolic pressure of the pulmonary artery, suggesting a balance between pre- and post-load.

One interesting aspect of this study is that during exercise with 60% of the maximum load, the variation in blood pressure levels (measured invasively) was of a small magnitude (Figure 1). Similar results occurred in another study that enrolled patients with severe heart failure (HF) who did not present hemodynamic instability during resistance exercises of the upper and lower limbs. In this individuals with HF class III and IV (EF: $25 \pm 2\%$, VO_{2peak} : $12.4 \pm 0.7 \text{ ml.kg}^{-1}.\text{min}^{-1}$), there was a slight decrease in the stroke volume with lower limb exercises, and maintenance of the stroke volume during upper limb exercises (relative to resting values), while the value of the CO was unchanged during both exercises.

The mechanisms that afford maintenance of ventricular function in healthy individuals, in ischemic cardiomyopathy patients, and in patients from different HF classes are not fully elucidated. Even so, it is important to note that, although some studies have demonstrated an increase in inotropism, the chronotropic response seems to be the main factor responsible for maintaining the CO¹⁹⁻²¹.

The typical behavior of increased systemic vascular resistance caused by pure isometric contraction²² many times

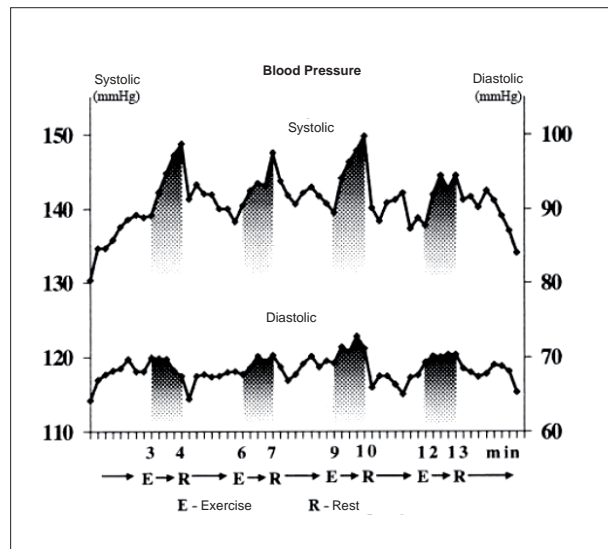


Fig. 1 - Temporal sequence of BP variation in HF patients during four sets of resistance exercise at 60% of the maximum load. Each series of exercises lasted for one minute and was followed by intervals (rest) that lasted for two minutes. Blood pressure was recorded every 15 seconds. The most significant increases occurred during the initial 15 to 30 seconds of the series, while the most significant reduction occurred in the first 15 to 30 seconds after the end of the exercise. Adapted from Meyer et al¹⁹.

is not desirable for cardiac patients. Nonetheless, contrary to what happens in isometric contractions, vascular resistance in cardiac patients is not elevated during the performance of a resistance exercise series (Figure 2)^{19-21,23}. This effect is possibly brought about by the dynamic component which is also present in the contractions of this type of exercise.

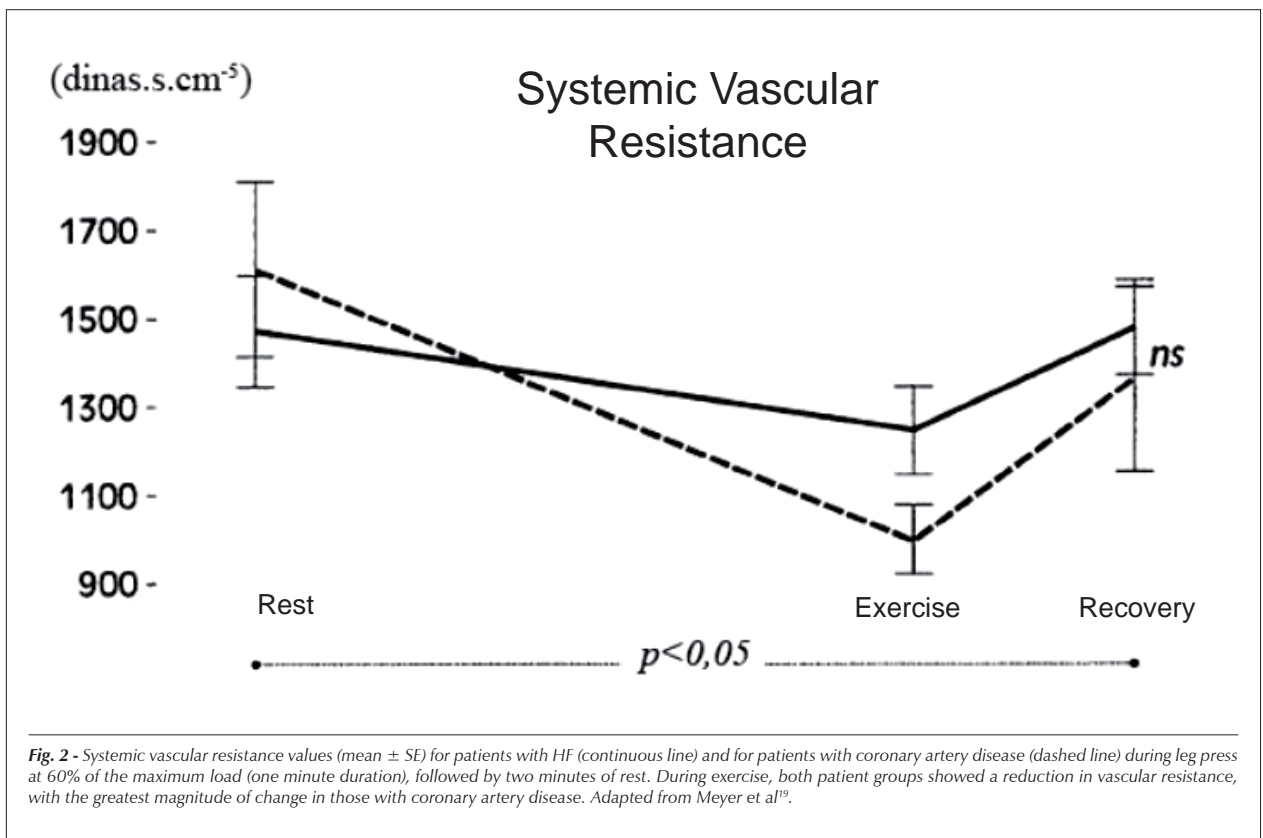
Resistance exercise and blood pressure

Regular physical exercise helps in the short-term and long-term control of blood pressure (BP) and is therefore indicated as coadjuvant intervention in managing hypertension²⁴. For a wider discussion of the effects of exercise on BP, it is important to note that this may be influenced not only by adaptations resulting from chronic physical training (chronic adaptations), but also by the effects of a single exercise session (subacute or post-exercise effects).

Resistance training: Chronic effects

More recent evidence does not support the ancient dogma that resistance training could elevate resting BP. This reasoning originated from the idea that greater pressure gains associated with resistance exercise (due to the greater isometric component) would lead to a chronic elevation of the BP.

Two robust meta-analyses demonstrated beneficial effects of resistance training on resting SAP and DAP. Both reviews included studies with the following characteristics: 1) randomized studies using a control group without exercise; 2) resistance training as a single intervention; 3) previously sedentary normotensive or hypertensive individuals; and 4) a minimum duration of four weeks. Kelley and Kelley²⁵ examined 11 studies, with a total of 320 subjects (182 with exercise and 138 controls), and found a reduction in SAP



(-3 ± 3 mmHg; 95% Confidence Interval: -4 to -1 mmHg) and DAP (-3 ± 2 mmHg; 95% Confidence Interval: -4 to -1 mmHg), which represented an approximate drop of 2% and 4%, respectively.

Posteriorly, a meta-analysis published by Cornelissen and Fagard²⁶, composed by 12 studies and 341 participants, showed the same tendency with reductions in SAP (-3.2 mmHg; $p=0.10$) and DAP (-3.5 mmHg; $p<0.01$) for the individuals exposed to resistance training. When the analyses of subgroups were carried out, again there were no differences related to the intensity of exercise, as well as when conventional or circuit resistance training was used. In conventional training, all sets of a certain movement are executed before initiating the following exercise, and there are normally higher loads and longer intervals than in circuit training.

On the other hand, in this other training mode, activity is more continuous and intervals are shorter since only one series is executed at each station, with immediate progression to the next station, and the circuit is repeated more times if necessary. Even if resistance training, similar to aerobic training²⁷, provides merely mild changes in the BP values, in terms of the population, this can have an impact in terms of a lower incidence of coronary disease and strokes²⁸.

Hypotension after resistance exercise

Physical exercise can promote a sustained drop in post-exercise blood pressure levels. Called post-exercise hypotension (PEH), this classic effect has been well

demonstrated with aerobic exercise²⁹ and has been studied in strength training both in ambulatory 24-hour evaluations (ABPM) and assessments made during the period of recovery (up to approximately 90 minutes after exercise). Bermudes et al³⁰ did not observe differences in 24-hour SAP and DAP values after one control session without exercise, and after one session of circuit resistance exercises. However, during the sleep period, there was a significant drop in BP after the resistance exercise session in comparison with the period after the control condition. Likewise, in young individuals with different levels of physical conditioning – sedentary, resistance-trained, and endurance-trained. SAP, DAP, and MAP were not different after one session of exercise or one control session³¹. Very recently, when studying 20 hypertensive women who use an angiotensin-converting enzyme inhibitor (captopril), Melo et al³¹ noted a significant reduction in blood pressure (SAP: -12 ± 3 mmHg, DAP: -6 ± 2 mmHg) during the recovery period up to 120 minutes after a low-intensity resistance exercise session. Additionally, the values observed by ABPM remained significantly lower for up to 10 hours after exercise (during the awake period), which did not occur after the control session³².

According to what is illustrated in Figure 3, in resistance exercise, PEH is more accentuated during recovery (≈ 90 minutes after the session), and tends to return to basal levels in the subsequent hours. Even though this effect was verified in normotensive and hypertensive individuals³¹⁻³⁴, controversy still exists, especially related to the intensity of the exercise. Some authors have documented a reduction in SAP after

Review Article

sessions using high loads^{35,36}; in other studies, however, high-intensity protocols were not effective in promoting significant pressure reductions³⁷⁻³⁹.

Recently, Rezk et al³⁵ demonstrated that sessions at 40% and 80% of the maximum load were followed by a reduction in SAP during the recovery (-6 ± 1 mmHg and -8 ± 1 mmHg, $p < 0.05$, respectively), while the DAP was reduced only after the lowest intensity session (40% of the maximum load). According to the authors, the hypotension generated by both protocols was mediated by the lower CO, and only modest elevations were noted in both systemic vascular resistance and post-exercise HR³⁵. Based on the abovementioned data, it is clear that resistance exercise performed chronically or acutely does not seem to offer risks as to an increase in BP, and this intervention may facilitate long-term pressure control.

Influence of resistance training in arterial compliance: emerging evidence

The distension capacity of arteries in response to differences in intravascular pressure (arterial compliance) enables the reduction of pressure fluctuations in the central circulation and contributes towards an adequate direction of flow. In sedentary elderly individuals, increased arterial stiffness is noted (the quality opposite of arterial compliance), which is also increased in pathological conditions such as atherosclerosis, insulin resistance, and diabetes mellitus⁴⁰. Another independent risk factor for increased arterial stiffness is obesity⁴¹. Moreover, aortic stiffness is a predictor of cardiovascular mortality and all-cause mortality in hypertensive patients⁴². Since regular exercise has a healthy impact on several risk factors associated with arterial stiffness, some studies have tested this intervention to possible changes in the distensibility parameters.

Observational and interventional studies have shown that regular aerobic exercise seems to attenuate age-related arterial stiffening and contribute to an increase in arterial compliance after systematic physical training⁴³⁻⁴⁶. Thus, over the last years, interest in investigating possible benefits of resistance exercise on vascular distensibility has grown, but evidence has not yet confirmed this hypothesis. Cross-sectional studies have noted less arterial compliance in adults from different age groups who are resistance-trained when compared to their sedentary peers^{47,48}. In young women, Cortez-Cooper et al verified that 11 weeks of high-intensity resistance training led to an elevation of central arterial stiffness (an increase in the augmentation index of systolic in the carotid artery and in the aortic pulse wave velocity)⁴⁹.

In an elegant study design with duration of 8 months, Miyachi et al randomized young men in control and intervention groups; high-intensity resistance training (80% of the maximum load) was used for four months, followed by a period of detraining (no exercise) in the four subsequent months. At the end of the training period, reduced compliance ($p < 0.01$) and increased arterial stiffness ($p < 0.01$) were noted, with significant correlations between the changes in carotid compliance and mass indexes ($r = -0.56$, $p < 0.001$) and left ventricular hypertrophy ($r = -0.68$, $p < 0.001$). After the

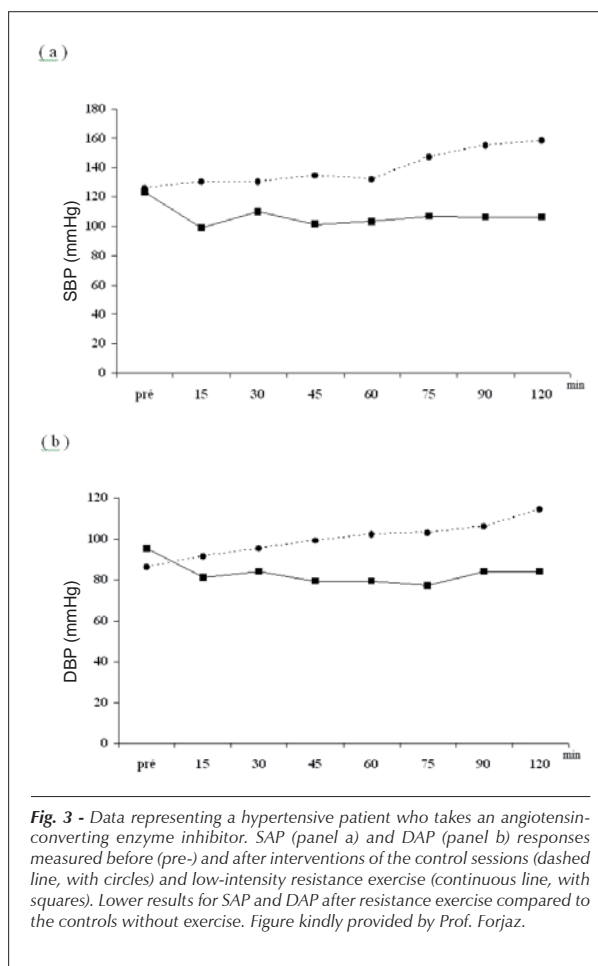


Fig. 3 - Data representing a hypertensive patient who takes an angiotensin-converting enzyme inhibitor. SAP (panel a) and DAP (panel b) responses measured before (pre-) and after interventions of the control sessions (dashed line, with circles) and low-intensity resistance exercise (continuous line, with squares). Lower results for SAP and DAP after resistance exercise compared to the controls without exercise. Figure kindly provided by Prof. Forjaz.

detraining, the variables modified by exercise returned to the basal (pre-study) values⁵⁰.

In contrast, another study that also enrolled young men who were exposed to three months of training with slightly lower volume and intensity sessions, showed no changes in arterial stiffening and cardiac dimensions⁵¹. Another very recently published study using moderate-intensity resistance training (50% of the maximum load, four months of training) showed a decrease in arterial compliance ($p < 0.01$) and in increase in the arterial stiffness index ($p < 0.01$), alterations that disappeared after four subsequent months without training. Nevertheless, for the group that performed a combined training (resistance training at 80% of the maximum load plus 30 minutes of aerobic exercise at the end of the session), there was no arterial stiffening; on the contrary, this type of training tended to increase compliance⁵².

Based on these findings, it is important to mention that the mechanisms that explain temporary arterial hardening observed in most studies after the exclusive use of resistance training are not yet known. The currently adopted hypothesis seems to tend towards changes in the structural content of arteries (elastin and collagen).

If considered alone, the information described here could suggest potential ill effects of resistance training, but

these effects do not seem to supplant the other adaptations afforded by resistance training. Therefore, these observations should not serve as a contraindication for this type of exercise. For example, a very recent report indicated that elderly individuals, who usually have greater arterial stiffness, did not demonstrate arterial hardening although they did show a reduction in central BP after 20 weeks of resistance training⁵³. Finally, even though in young men resistance exercise may seem to cause a decrease in compliance, this result seems to be perfectly neutralized by aerobic exercise.

Resistance training and its implications for vascular function and basal blood flow

The loss or attenuation of physiological endothelium-mediated vasodilation – endothelial dysfunction – is an early event in the atherosclerotic process and it is associated with several risk factors in adults and children^{54,55}. In the presence of HF (ischemic or non-ischemic), endothelial dysfunction is associated with increased mortality⁵⁶. On the other hand, some evident findings have helped to establish improved endothelial function and increased blood flow in HF patients after aerobic training^{57,58}. This vascular adaptation seems to also occur after resistance exercise programs associated with aerobic stimuli. Maiorana et al⁵⁹ noted an increase in dependent and independent endothelial vasodilation in HF patients (EF: $26 \pm 3\%$, VO_2^{peak} : $19.5 \pm 1.2 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$) after eight weeks of combined training⁵⁹. In cardiac patients with coronary disease, combined training predominantly of the lower limbs increased flow-mediated dilation by the flow in the brachial artery, indicating systemic endothelial adaptation afforded by exercise⁶⁰, whereas the response independent of the endothelium was unchanged.

As to exclusive resistance training, there is evidence that clearly shows its effects primarily on the increase of basal blood flow. Initially, a pilot study showed that after 11 weeks of circuit training, patients with HF class II and III (EF: $26 \pm 6\%$) obtained an increase in resting blood flow ($p < 0.01$), albeit without changes in vasodilation responses (endothelial response)⁶¹. Nonetheless, another study evidenced results such as an increased basal blood flow besides an improvement in endothelial function⁶². In this randomized controlled clinical study, the HF patients submitted to three months of moderate-intensity resistance training showed an increase in blood flow, both at rest and in response to exercise stimuli or ischemia (greater hyperemia after occlusion, indicating an increase in vasodilation mediated by the endothelium).

Resistance training also seems to elevate the basal blood flow of healthy individuals. In healthy young men who underwent three months of resistance training, an increase in resting blood flow was noted in addition to evidence of arterial remodeling, findings observed from the increased diameter of the brachial artery. However, this sample showed no changes in brachial endothelial function. The explanation for these findings may be related to the fact that participants had normal vascular function at the onset of the study, which would not provide a greater adaptation during the three months of training⁶³.

On the other hand, in aging, blood flow and vascular conductance diminish, and this can contribute to a reduction in muscle perfusion and functional capacity⁶⁴. While aerobic exercise does not seem to attenuate the drop in blood flow that happens with aging, a cross-sectional study suggested that men submitted to strength training did not display a decrease in age-related peripheral blood flow^{65,66}. Based on this, data from a recent prospective study indicated the apparent efficacy of resistance training on the maintenance of peripheral blood flow in individuals with advanced ages⁶⁷. In this paper, elderly and middle-aged adults randomized to resistance training for 13 weeks showed an increase in blood flow and vascular conductance in the femoral artery.

In summary, information available in medical literature indicates that resistance training, whether carried out exclusively or in combination with aerobic exercises, can improve endothelial function. Additionally, evidence also points to an important effect of resistance exercise on the increase of peripheral blood flow, which can contribute to minimizing the functional limitations present in aging or in different pathological conditions.

Final considerations

Resistance exercise has progressively gained importance in cardiovascular prevention and rehabilitation programs. The perspectives of possible studies with relevant validated outcomes may reinforce the current reliable information that allows safe prescriptions for the practice of this type of exercise by different populations.

The proposal of this review was to update the evidence concerning cardiovascular responses to chronic and acute resistance exercise. The information found in literature demonstrates hemodynamic stability during exercise and potential effects that help in blood pressure control. In addition, this review presents important peculiarities of arterial distensibility adaptation, vascular function, and peripheral blood flow in face of resistance training.

Finally, the body of evidence available on the applicability and safety of resistance training in cardiac patients is consistent with what is already established regarding aerobic exercise.

Potential Conflict of Interest

No potential conflict of interest relevant to this article was reported.

Sources of Funding

There were no external funding sources for this study.

Study Association

This article is part of the thesis of master submitted by Daniel Umpierre, from Universidade Federal do Rio Grande do Sul (UFRGS), Laboratório de Fisiopatologia do Exercício (LasiEx), Serviço de Cardiologia.

References

1. Humphreys PW, Lind AR. The blood flow through active and inactive muscles of the forearm during sustained hand-grip contractions. *J Physiol.* 1963; 166: 120-35.
2. Lind AR, McNicol GW. Circulatory responses to sustained hand-grip contractions performed during other exercise, both rhythmic and static. *J Physiol.* 1967; 92 (3): 595-607.
3. Sociedade Brasileira de Cardiologia. Diretriz de reabilitação cardíaca. *Arq Bras Cardiol.* 2005; 84 (5): 431-40.
4. Braith RW, Stewart KJ. Resistance exercise training: its role in the prevention of cardiovascular disease. *Circulation.* 2006; 113 (22): 2642-50.
5. Bjarnason-Wehrens B, Mayer-Berger W, Meister ER, Baum K, Hambrecht R, Gielen S. Recommendations for resistance exercise in cardiac rehabilitation. Recommendations of the German Federation for Cardiovascular Prevention and Rehabilitation. *Eur J Cardiovasc Prev Rehabil.* 2004; 11 (4): 352-61.
6. American Heart Association. Resistance exercise in individuals with and without cardiovascular disease: benefits, rationale, safety, and prescription: an advisory from the Committee on Exercise, Rehabilitation, and Prevention, Council on Clinical Cardiology, American Heart Association; Position paper endorsed by the American College of Sports Medicine. *Circulation.* 2000; 101 (7): 828-33.
7. Hunter GR, Treuth MS, Weinsier RL, Kekes-Szabo T, Kell SH, Roth DL, et al. The effects of strength conditioning on older women's ability to perform daily tasks. *J Am Geriatr Soc.* 1995; 43 (7): 756-60.
8. Hickson RC, Rosenkoetter MA, Brown MM. Strength training effects on aerobic power and short-term endurance. *Med Sci Sports Exerc.* 1980; 12 (5): 336-9.
9. Ades PA, Ballor DL, Ashikaga T, Utton JL, Nair KS. Weight training improves walking endurance in healthy elderly persons. *Ann Intern Med.* 1996; 124 (6): 568-72.
10. Hunter GR, McCarthy JP, Bamman MM. Effects of resistance training on older adults. *Sports Med.* 2004; 34 (5): 329-48.
11. McCartney N, McKelvie RS, Martin J, Sale DG, MacDougall JD. Weight-training-induced attenuation of the circulatory response of older males to weight lifting. *J Appl Physiol.* 1993; 74 (3): 1056-60.
12. Tanasescu M, Leitzmann MF, Rimm EB, Willett WC, Stampfer MJ, Hu FB. Exercise type and intensity in relation to coronary heart disease in men. *JAMA.* 2002; 288 (16): 1994-2000.
13. Forjaz CL, Rezk CC, Cardoso Jr CF. In: Negrão CE, Pereira Barreto AC (eds.). *Cardiologia do exercício: do atleta ao cardiopata.* São Paulo: Manole; 2005. p. 260-71.
14. Fleck SJ, Dean LS. Resistance-training experience and the pressor response during resistance exercise. *J Appl Physiol.* 1987; 63 (1): 116-20.
15. Lewis SF, Snell PG, Taylor WF, Hamra M, Graham RM, Pettinger WA, et al. Role of muscle mass and mode of contraction in circulatory responses to exercise. *J Appl Physiol.* 1985; 58 (1): 146-51.
16. MacDougall JD, Tuxen D, Sale DG, Moroz JR, Sutton JR. Arterial blood pressure response to heavy resistance exercise. *J Appl Physiol.* 1985; 58 (3): 785-90.
17. Werber-Zion G, Goldhammer E, Shaar A, Pollock ML. Left ventricular function during strength testing and resistance exercise in patients with left ventricular dysfunction. *J Cardiopulm Rehabil.* 2004; 24 (2): 100-9.
18. Gordon NF, Kohl HW, Pollock ML, Vaandrager H, Gibbons LW, Blair SN. Cardiovascular safety of maximal strength testing in healthy adults. *Am J Cardiol.* 1995; 76 (11): 851-3.
19. Meyer K, Hajric R, Westbrook S, Haag-Wildi S, Holtkamp R, Leyk D, et al. Hemodynamic responses during leg press exercise in patients with chronic congestive heart failure. *Am J Cardiol.* 1999; 83 (11): 1537-43.
20. Cheatham C, Green D, Collis J, Dembo L, O'Driscoll G. Effect of aerobic and resistance exercise on central hemodynamic responses in severe chronic heart failure. *J Appl Physiol.* 2002; 93 (1): 175-80.
21. McKelvie RS, McCartney N, Tomlinson C, Bauer R, MacDougall JD. Comparison of hemodynamic responses to cycling and resistance exercise in congestive heart failure secondary to ischemic cardiomyopathy. *Am J Cardiol.* 1995; 76 (12): 977-9.
22. Elkayam U, Roth A, Weber L, Hsueh W, Nanna M, Freidenberger L, et al. Isometric exercise in patients with chronic advanced heart failure: hemodynamic and neurohumoral evaluation. *Circulation.* 1985; 72 (5): 975-81.
23. King ML, Dracup KA, Fonarow GC, Woo MA. The hemodynamic effects of isotonic exercise using hand-held weights in patients with heart failure. *J Heart Lung Transplant.* 2000; 19 (12): 1209-18.
24. Chobanian AV, Bakris GL, Black HR, Cushman WC, Green LA, Izzo JL Jr, et al. The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure: the JNC 7 report. *JAMA.* 2003; 289 (19): 2560-72.
25. Kelley GA, Kelley KS. Progressive resistance exercise and resting blood pressure: a meta-analysis of randomized controlled trials. *Hypertension.* 2000; 35 (3): 838-43.
26. Cornelissen VA, Fagard RH. Effect of resistance training on resting blood pressure: a meta-analysis of randomized controlled trials. *J Hypertens.* 2005; 23 (2): 251-9.
27. Whelton SP, Chin A, Xin X, He J. Effect of aerobic exercise on blood pressure: a meta-analysis of randomized, controlled trials. *Ann Intern Med.* 2002; 136 (7): 493-503.
28. Whelton PK, He J, Appel LJ, Cutler JA, Havas S, Kotchen TA, et al. Primary prevention of hypertension: clinical and public health advisory from The National High Blood Pressure Education Program. *JAMA.* 2002; 288 (15): 1882-8.
29. Kenney MJ, Seals DR. Postexercise hypotension: key features, mechanisms, and clinical significance. *Hypertension.* 1993; 22 (5): 653-64.
30. Bermudes AM, Vassallo DV, Vasquez EC, Lima EG. Ambulatory blood pressure monitoring in normotensive individuals undergoing two single exercise sessions: resistive exercise training and aerobic exercise training. *Arq Bras Cardiol.* 2004; 82 (1): 57-64.
31. Melo CM, Alencar Filho AC, Tinucci T, Mion Jr D, Forjaz CL. Postexercise hypotension induced by low-intensity resistance exercise in hypertensive women receiving captopril. *Blood Press Monit.* 2006; 11 (4): 183-9.
32. MacDonald JR, MacDougall JD, Interisano SA, Smith KM, McCartney N, Moroz JS, et al. Hypotension following mild bouts of resistance exercise and submaximal dynamic exercise. *Eur J Appl Physiol Occup Physiol.* 1999; 79 (2): 148-54.
33. Fisher MM. The effect of resistance exercise on recovery blood pressure in normotensive and borderline hypertensive women. *J Strength Cond Res.* 2001; 15 (2): 210-6.
34. Mediano MFF, Paravidino V, Simão R, Pontes FL, Polito MD. Subacute behaviour of the blood pressure after power training in controlled hypertensive individuals. *Braz J Sport Med.* 2005; 11 (6): 347-40.
35. Rezk CC, Marrache RC, Tinucci T, Mion D Jr, Forjaz CL. Post-resistance exercise hypotension, hemodynamics, and heart rate variability: influence of exercise intensity. *Eur J Appl Physiol.* 2006; 98 (1): 105-12.
36. Simao R, Fleck SJ, Polito M, Monteiro W, Farinatti P. Effects of resistance training intensity, volume, and session format on the postexercise hypotensive response. *J Strength Cond Res.* 2005; 19 (4): 853-8.
37. Raglin JS, Turner PE, Eksten F. State anxiety and blood pressure following 30 min of leg ergometry or weight training. *Med Sci Sports Exerc.* 1993; 25(9): 1044-8.
38. Focht BC, Koltyn KF. Influence of resistance exercise of different intensities on state anxiety and blood pressure. *Med Sci Sports Exerc.* 1999; 31 (3): 456-63.
39. O'Connor PJ, Bryant CX, Veltri JP, Gebhardt SM. State anxiety and ambulatory blood pressure following resistance exercise in females. *Med Sci Sports Exerc.* 1993; 25 (4): 516-21.

40. Safar ME, Levy BI, Struijker-Boudier H. Current perspectives on arterial stiffness and pulse pressure in hypertension and cardiovascular diseases. *Circulation*. 2003; 107 (22): 2864-9.
41. Zebekakis PE, Nawrot T, Thijs L, Balkestein EJ, van der Heijden-Spek J, Van Bortel LM, et al. Obesity is associated with increased arterial stiffness from adolescence until old age. *J Hypertens*. 2005; 23 (10): 1839-46.
42. Laurent S, Boutouyrie P, Asmar R, Gautier I, Laloux B, Guize L, et al. Aortic stiffness is an independent predictor of all-cause and cardiovascular mortality in hypertensive patients. *Hypertension*. 2001; 37 (5): 1236-41.
43. Vaitkevicius PV, Fleg JL, Engel JH, O'Connor FC, Wright JC, Lakatta LE, et al. Effects of age and aerobic capacity on arterial stiffness in healthy adults. *Circulation*. 1993; 88 (4): 1456-62.
44. Tanaka H, DeSouza CA, Seals DR. Absence of age-related increase in central arterial stiffness in physically active women. *Arterioscler Thromb Vasc Biol*. 1998; 18 (1): 127-32.
45. Cameron JD, Dart AM. Exercise training increases total systemic arterial compliance in humans. *Am J Physiol*. 1994; 266 (2): H693-701.
46. Tanaka H, Dinverno FA, Monahan KD, Clevenger CM, DeSouza CA, Seals DR. Aging, habitual exercise, and dynamic arterial compliance. *Circulation*. 2000; 102 (11): 1270-5.
47. Bertovic DA, Waddell TK, Gatzka CD, Cameron JD, Dart AM, Kingwell BA. Muscular strength training is associated with low arterial compliance and high pulse pressure. *Hypertension*. 1999; 33 (6): 1385-91.
48. Miyachi M, Donato AJ, Yamamoto K, Takahashi K, Gates PE, Moreau KL, et al. Greater age-related reductions in central arterial compliance in resistance-trained men. *Hypertension*. 2003; 41 (1): 130-5.
49. Cortez-Cooper MY, DeVan AE, Anton MM, Farrar RP, Beckwith KA, Todd JS, et al. Effects of high intensity resistance training on arterial stiffness and wave reflection in women. *Am J Hypertens*. 2005; 18 (7): 930-4.
50. Miyachi M, Kawano H, Sugawara J, Takahashi K, Hayashi K, Yamazaki K, et al. Unfavorable effects of resistance training on central arterial compliance: a randomized intervention study. *Circulation*. 2004; 110 (18): 2858-63.
51. Rakobowchuk M, McGowan CL, de Groot PC, Bruinsma D, Hartman JW, Phillips SM, et al. Effect of whole body resistance training on arterial compliance in young men. *Exp Physiol*. 2005; 90 (4): 645-51.
52. Kawano H, Tanaka H, Miyachi M. Resistance training and arterial compliance: keeping the benefits while minimizing the stiffening. *J Hypertens*. 2006; 24 (9): 1753-9.
53. Taaffe DR, Galvao DA, Sharman JE, Coombes JS. Reduced central blood pressure in older adults following progressive resistance training. *J Hum Hypertens*. 2007; 21 (1): 96-8.
54. Widlansky ME, Gokce N, Keaney JF Jr, Vita JA. The clinical implications of endothelial dysfunction. *J Am Coll Cardiol*. 2003; 42 (7): 1149-60.
55. Celermajer DS, Sorensen KE, Gooch VM, Spiegelhalter DJ, Miller OI, Sullivan ID, et al. Non-invasive detection of endothelial dysfunction in children and adults at risk of atherosclerosis. *Lancet*. 1992; 340 (8828): 1111-5.
56. Katz SD, Hryniewicz K, Hriljac I, Balidemaj K, Dimayuga C, Hudaihed A, et al. Vascular endothelial dysfunction and mortality risk in patients with chronic heart failure. *Circulation*. 2005; 111 (3): 310-4.
57. Hambrecht R, Fiehn E, Weigl C, Gielen S, Hamann C, Kaiser R, et al. Regular physical exercise corrects endothelial dysfunction and improves exercise capacity in patients with chronic heart failure. *Circulation*. 1998; 98 (24): 2709-15.
58. Roveda F, Middlekauff HR, Rondon MU, Reis SF, Souza M, Nastari L, et al. The effects of exercise training on sympathetic neural activation in advanced heart failure: a randomized controlled trial. *J Am Coll Cardiol*. 2003; 42 (5): 854-60.
59. Maiorana A, O'Driscoll G, Cheetham C, Collis J, Goodman C, Rankin S, et al. Combined aerobic and resistance exercise training improves functional capacity and strength in CHF. *J Appl Physiol*. 2000; 88 (5): 1565-70.
60. Walsh JH, Bilsborough W, Maiorana A, Best M, O'Driscoll GJ, Taylor RR, et al. Exercise training improves conduit vessel function in patients with coronary artery disease. *J Appl Physiol*. 2003; 95 (1): 20-5.
61. Hare DL, Ryan TM, Selig SE, Pellizzer AM, Wrigley TV, Krum H. Resistance exercise training increases muscle strength, endurance, and blood flow in patients with chronic heart failure. *Am J Cardiol*. 1999; 83 (12): 1674-7.
62. Selig SE, Carey MF, Menzies DC, Patterson J, Geerling RH, Williams AD, et al. Moderate-intensity resistance exercise training in patients with chronic heart failure improves strength, endurance, heart rate variability, and forearm blood flow. *J Card Fail*. 2004; 10 (1): 21-30.
63. Rakobowchuk M, McGowan CL, de Groot PC, Hartman JW, Phillips SM, MacDonald MJ. Endothelial function of young healthy males following whole body resistance training. *J Appl Physiol*. 2005; 98 (6): 2185-90.
64. Dinverno FA, Jones PP, Seals DR, Tanaka H. Limb blood flow and vascular conductance are reduced with age in healthy humans: relation to elevations in sympathetic nerve activity and declines in oxygen demand. *Circulation*. 1999; 100 (2): 164-70.
65. Dinverno FA, Seals DR, DeSouza CA, Tanaka H. Age-related decreases in basal limb blood flow in humans: time course, determinants and habitual exercise effects. *J Physiol*. 2001; 531 (Pt 2): 573-9.
66. Miyachi M, Tanaka H, Kawano H, Okajima M, Tabata I. Lack of age-related decreases in basal whole leg blood flow in resistance-trained men. *J Appl Physiol*. 2005; 99(4): 1384-90.
67. Anton MM, Cortez-Cooper MY, DeVan AE, Neidre DB, Cook JN, Tanaka H. Resistance training increases basal limb blood flow and vascular conductance in aging humans. *J Appl Physiol*. 2006; 101 (5): 1351-5.