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Cardiac autonomic responses during upper versus lower limb resistance exercise in healthy elderly men

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ABSTRACT | Objective: To investigate the cardiac autonomic responses during upper versus lower limb discontinuous resistance exercise (RE) at different loads in healthy older men. **Method:** Ten volunteers (65±1.2 years) underwent the one-repetition maximum (1RM) test to determine the maximum load for the bench press and the leg press. Discontinuous RE was initiated at a load of 10%1RM with subsequent increases of 10% until 30%1RM, followed by increases of 5%1RM until exhaustion. Heart rate (HR) and R-R interval were recorded at rest and for 4 minutes at each load applied. Heart rate variability (HRV) was analyzed in 5-min segments at rest and at each load in the most stable 2-min signal. **Results**: Parasympathetic indices decreased significantly in both exercises from 30%1RM compared to rest (rMSSD: 20±2 to 11±3 and 29±5 to 12±2 ms; SD1: 15±2 to 8±1 and 23±4 to 7±1 ms, for upper and lower limb exercise respectively) and HR increased (69±4 to 90±4 bpm for upper and 66±2 to 89±1 bpm for lower). RMSM increased for upper limb exercise, but decreased for lower limb exercise (28±3 to 45±9 and 34±5 to 14±3 ms, respectively). In the frequency domain, the sympathetic (LF) and sympathovagal balance (LF/HF) indices were higher and the parasympathetic index (HF) was lower for upper limb exercise than for lower limb exercise from 35% of 1RM. **Conclusions**: Cardiac autonomic change occurred from 30% of 1RM regardless of RE limb. However, there was more pronounced sympathetic increase and vagal decrease for upper limb exercise than for lower limb exercise. These results provide a basis for more effective prescription of RE to promote health in this population.

Keywords: physical therapy; resistance exercise; autonomic nervous system; elderly; upper limbs; lower limbs.

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Introduction

Resistance exercise (RE) training has become an important component of physical exercise programs for older people[1]. It is well established that RE training combined with endurance training is the most effective way to achieve the expected beneficial results of the physical training program[1,2].

Several studies have shown that strength (resistance) training can counteract age-related impairments[3,4]. Aging is associated with decreases in muscle mass, strength[5,6], and cardiovascular fitness and with increased cardiovascular risk factors. Some age-related chronic diseases include cardiovascular disease, type 2 diabetes and obesity[7]. Impaired heart rate variability (HRV) with increased sympathetic modulation and decreased parasympathetic modulation is also related to aging[8,9].

Considering all age-related physiological changes, an RE program may be able to enhance muscle strength, power, and local muscular endurance[10,11], increase HRV and, consequently, decrease the risk of mortality[8,12] and co-morbidities associated with advancing age[9,13]. Therefore, RE is a valuable tool for physical therapists and understanding the cardiovascular adjustments involved in it can provide the theoretical basis for better prescription of these exercises to effectively promote health in this population.

Cardiovascular responses to RE depend on exercise intensity and muscle mass[1]. In this context, upper limb RE generates different cardiovascular responses compared to lower limb exercise. It is well-established that upper limb exercise leads to higher blood pressure (BP) and heart rate (HR) than lower limb exercise due

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to a higher work component and elevated peripheral resistance caused by reduced active muscle mass[14-16]. In addition, upper limb exercise also induces greater perceived exertion compared to leg exercise at the same relative workload[17].

Regarding autonomic cardiac responses, Tulppo et al.[18] found a more rapid vagal withdrawal in arm cycle ergometer exercise than leg cycle ergometer exercise in young healthy men. This condition leads to higher HR and hemodynamic changes in arm exercise and it may be explained by a more powerful central command by vagal withdrawal and a stronger muscle afferent input in upper limb exercise.

However, the autonomic responses of upper versus lower limbs in RE are still unclear. Consequently, the aim of this study was to investigate the cardiac autonomic responses during upper versus lower limb discontinuous RE at different loads in healthy older men. The hypothesis was that the cardiac responses would be different.

Method

Subjects

Ten healthy elderly men were included in this study after clinical evaluation. The inclusion criteria were: age between 60 and 80 years; no participation in exercise training programs such as resistance or aerobic training in the previous 6 months; no obesity; and apparent good health. The exclusion criteria were: current smoking habit, current use of any medication, musculoskeletal pain or difficulty in understanding the RE protocol.

Screening of subjects was performed with clinical evaluation, physical evaluation, and laboratory tests, including analysis of hemoglobin, triglycerides, total cholesterol, low-density lipoprotein (LDL) and high-density lipoprotein (HDL), fasting glucose, and uric acid. For clinical safety, a physician-supervised exercise test with gas analysis (CPX-D, Medical Graphics, St. Paul, MN, USA) was performed for all volunteers before participation in our experimental protocol. The volunteers were considered able to perform the protocol when the cardiopulmonary, metabolic, and electrocardiographic variables showed normal physiological behavior during testing.

This study was approved by the Human Ethics Committee of Universidade Federal de São Carlos (196/2006), São Carlos, SP, Brazil, and followed the principles of the declaration of Helsinki. All subjects gave written informed consent for participation.

Procedures

After clinical evaluation, all volunteers underwent a physical and anthropometric evaluation. Height (m) and weight (kg) were measured and body mass index (BMI) (kg/m^2) was calculated using a scale and stadiometer (Welmy, São Paulo, SP, Brazil). The subsequent evaluations (maximum load determination and incremental RE) were performed with a one-week interval. The tests (upper or lower limb) were performed in a random order defined by numbered envelopes. All tests were performed within a three-week period.

Experiments were conducted at approximately the same time of day (morning) to minimize subject-to-subject variation due to circadian rhythms at a controlled room temperature of 22-24 °C and relative air humidity of 50-60%. Each volunteer was instructed to wear comfortable clothes for the test; to avoid alcoholic and caffeinated beverages 24 hours before the test; to avoid heavy meals at least 2 hours before the test; and to abstain from exhaustive training on the day prior to the test.

Immediately prior to data collection, subjects confirmed a normal night's sleep and HR and blood pressure were checked to ensure they were within the normal range. The subjects were instructed to avoid unnecessary talking during the protocol in order to minimize interference in the HR and eletrocardiogram (ECG) signal and to take note of symptoms before, during, and after the protocols.

One-repetition maximum (1RM) test – 45° Incline Bench Press and Leg Press

To determine the protocol loads for RE, the subjects were tested for maximal strength performing a 1RM test for upper limb using the incline bench press set at 45° (Vitally Convergent, São Paulo, Brazil) and for lower limbs using the leg press at 45° (Pro-Fitness, São Paulo, Brazil) with a one-week interval.

During the 1RM test on the incline bench press, the volunteers remained seated with trunk inclined at 45° from horizontal and feet on the ground. They were instructed to hold the free bar which supported the load. During exercise, the elbow and shoulder were flexed and then returned to extension as the initial position, avoiding isometric contraction and the Valsalva maneuver[19]. Based on a previous study[20], the 1RM load was estimated (1RM-E) by multiplying the body weight by 0.6.

For the 1RM test on the leg press at 45°, the volunteer remained seated with trunk inclined at 45° from horizontal and knees and hips flexed at 90°. During the exercise, the knees and hips were extended and returned to the initial position after the flexion position avoiding isometric contraction and the Valsalva maneuver[19]. Based on a previous study[21], the 1RM-E was estimated by multiplying the body weight by 4.

For both exercises, 80% was applied to the 1RM-E value to start the test. If the estimated load was not the maximum, an increase of 10% was achieved after 5 minutes or the time required for vital signs to return to baseline levels. It was expected that 1RM would be determined in up to six attempts[22].

Discontinuous incremental resistance exercise protocol

One week after the 1RM test, a discontinuous incremental RE protocol was performed with the same equipment and movements described for the maximum test. The volunteer remained at rest seated on the equipment for 10 min. After this, the protocol began at a load of 10%1RM and subsequent increases of 10% until 30%1RM. After that, the incremental adjustments were 5%1RM until exhaustion or SBP>200 mmHg, HR≥85% of maximum (HR (220 - age × 0.85)) and the development of any potential arrhythmias. The volunteers performed 4 min of exercise at a cadence of 12 repetitions per minute in each load combined with respiratory rhythm of 2 seconds of inspiration and 3 of expiration. Respiratory standardization was determined to reduce the influence of the respiratory rhythm on autonomic modulation, more specifically on high frequency bands. A recovery period of approximately 15 min was observed in order to return to base HR and BP values. The incremental tests (supine and leg) were also separated by a one-week interval.

Safety and ECG monitoring during protocols

During evaluation, HR in real time was verified and recorded by an HR monitor (Polar Vantage, Kempele, Finland) linked to an interface (Polar Advantage, Kempele, Finland) and a microcomputer (Notebook Soyo - PW 9800, Taiwan, China). In addition, an ECG monitor (Ecafix TC 500, São Paulo, Brazil) was used to detect any potential arrhythmias or ischemia. Blood pressure was verified by auscultation (sphygmomanometer BD, São Paulo, Brazil) before and after each test and pain and muscle fatigue were evaluated using the Borg scale at end of each stage[23].

Heart rate and R-R interval data acquisition, signal processing and HRV analysis

HR and R-R interval (R-Ri) were recorded using an HR monitor (Polar Electro™, Kempele, Finland). Measurements were subsequently obtained at rest before starting the exercise on the respective bench press or leg press at 45° for 10 min and during incremental RE exercise protocol for 4 minutes.

After acquisition, the signals were transferred to the Polar Precision Performance Software and visually inspected and corrected for ectopic beats (premature, supraventricular, and ventricular). Times series data were processed using the Kubios HRV Analysis software (MATLAB, version 2 beta, Kuopio, Finland).

For HRV analysis, we used the HR and R-Ri signals acquired in the pre-exercise rest period and during exercise. The segments selected were 5 minutes of rest and visually the most stable exercise section containing 2 min free of noise at each load effort, excluding the initial 40 seconds of the exercise (period during which a rapid withdrawal of vagal activity occurred) and the final 30 seconds to avoid influences of blood pressure measurement.

The HRV indices obtained were rMSSD, RMSM, SD1, LF, and HF. The rMSSD consists of the square root of the mean of the sum of the squares of the differences between the R-Ri registered, divided by the number of R-Ri in a specified time period minus one, which provided information on parasympathetic cardiac modulation[24]. The RMSM index corresponds to the square root of the sum of the squares of the differences of the individual values in relation to the mean value, divided by the number of R-Ri in a specified time period, characterized as a marker of the total HRV[25].

Quantitative Poincaré plot analysis consisted of plotting each R-Ri as a function of the preceding interval and in this type of analysis the stationary signal is not required. The SD1 index was obtained in milliseconds (ms), which provides information on the standard deviation of instantaneous beat-to-beat variability, characterized as a parasympathetic cardiac modulation marker[26].

Frequency-domain analysis of HRV was performed using Fast Fourier Transformation[27]. Two spectral components were obtained: low frequency (LF), from 0.04 to 0.15 Hz, and high frequency (HF), from 0.15 to 0.4 Hz. The spectral components were expressed in normalized units (nu) and also in absolute units (ms^2) to HF. Normalization is obtained by dividing the absolute power of a spectral component (LF or HF component) by the total power minus the power of the component, with a frequency range between 0 and 0.03 Hz (very low frequency), and by multiplying this ratio by 100[28].

Statistical analysis

The sample size was calculated using the GraphPad StatMate software, version 1.01. To reach statistical significance (p<0.05) at a power of 80%, a sample of 8 subjects was required to demonstrate a mean difference between upper and lower limbs, based on the rMSSD index in a previous study[21].

Data normality was verified by the Shapiro-Wilk test, and the data were expressed in mean and standard error of the mean (SEM). Student's *t*-test was used to compare the 1RM test between upper and lower limb at rest and at the end of exercise. Repeated measures analysis of variance (ANOVA) was used to compare the variables at different loads of 1RM. Tukey-Kramer *post-hoc* was used to identify differences. Analysis was performed using the software STATISTICA for Windows (Stat Soft Inc., 2000). A p value less than 0.05 was taken to denote statistical significance.

Results

Ten healthy, previously sedentary older adults successfully completed the proposed protocol. Descriptive characteristics of participants are: 10 males, age 65.2±1.2 years, weight 70.0±2.2 kg, height 1.66±0.01 m, BMI 25.0±0.8 kg/m2. There were no adverse events experienced due to either exercise. The exercise interruption criterion reported by all volunteers was muscle fatigue.

Cardiovascular variables and HRV indices in the upper and lower limb exercises (1RM test and incremental resistance exercise protocol) are shown in Table 1. For the 1RM test, there was a significant increase in HR when comparing rest and the peak of exercise, however, no difference was found between the different exercise modalities.

In relation to the incremental resistance exercise protocol, there was a significantly higher value in SBP and HR when comparing rest and peak of exercise

Table 1. Cardiovascular variables and HRV in upper and lower limb exercises at rest and peak of 1RM test and incremental resistance exercise (RE) protocol in healthy older men.

	Upper limb		Lower limb	
	Rest	**Peak**	**Rest**	**Peak**
1RM test				
SBP (mmHg)	124±3	138±2	131±2	141±2
DBP (mmHg)	76±1	77±1	81±1	85±1
HR (mmHg)	69±4	90±4*	66±2	89±1*
Borg Scale (0-10)	-	7±1	-	9±0.1
Load 1RM (kg)	-	43±2	-	316±68.0
Incremental RE				
SBP (mmHg)	119±4	140±4*	123±3	167±7*§
DBP (mmHg)	75±2	79±2	78±2	89±4*
HR (bpm)	69±3	93±6*	65±3	92±4*
Borg Scale (0-10)	-	8±1	-	8±1
Load RE (kg)	-	19±1	-	153±14
HRV indices				
rMSSD (ms)	20±2	11±3*	29±5	12±2*
RMSM (ms)	28±3	45±9*	34±5	14±3*§
SD1 (ms)	15±2	8±1*	23±4	7±1*
LF (n.u.)	67±5	91±2*	66±6	81±2§
HF (n.u.)	31±5	8±2*	31±6	18±3
LF/HF (n.u.)	3±0.5	16±4*	3±1	5±1
HF (ms^2)	139±85	33±21*	188±124	97±63*§

Data as mean±standard error of mean (SEM). SBP: systolic blood pressure; DBP: diastolic blood pressure; HR: heart rate; HRV: heart rate variability; rMSSD: root mean square of successive differences; RMSM: root mean square of the differences from the mean interval; SD1: standard deviation of the instantaneous R-R interval; LF: low frequency; HF: high frequency; n.u.: normalized units. *p<0.05 compared to rest condition; § p<0.05 compared upper and lower limb exercise (one-way ANOVA with repeated measures).

in both upper and lower limb exercises, while for diastolic blood pressure (DBP), this difference was observed only for exercise performed on the leg press. In addition, a significantly higher SBP was found at the end of lower limb exercise compared to upper limb exercise.

Regarding cardiac autonomic responses, Table 1 shows the HRV indices obtained at rest and peak of discontinuous incremental exercise protocol. For both groups, there was a significant decrease in rMSSD, SD1, and HF in absolute units (ms^2) comparing rest and peak of exercise. For the RMSM index, however, there was an increase for the incline bench press and a decrease for the leg press at 45°.

For upper limb exercise, there was a significant increase in LF and LF/HF and a decrease in HF also comparing rest and peak of exercise for these frequency-domain indices assessed in normalized units. Additionally, at the peak of incremental load, intergroup differences were also observed with significantly higher values for RMSM and LF (nu) and lower values for HF (ms^2) in upper limb exercises compared to lower limb exercises.

Considering the results on different % loads during the incremental protocol, we observed a significant decrease in the rMSSD index (Figure 1A) and SD1 index (Figure 1B) from 30%1RM compared to rest, while the HR values increased from this percentage (Figure 1D). In contrast, the RMSM index decreased in lower limb exercise and increased in upper limb from 30%1RM. From this load, there was a significant difference between exercise modalities, with higher values for the incline bench press (Figure 1C).

The behavior of the frequency-domain indices of HRV are shown in Figure 2. The LF component (Figure 2A) increased and HF (Figure 2B) decreased significantly from 30%1RM in the incline bench press. Regarding the LF/HF ratio (Figure 2C), there was a significant increase from 35% of 1RM in the bench press. In addition, we found differences between exercises at 35% and 40% of 1RM, with higher values of LF and lower HF in the upper limb exercise. For LF/HF, this difference was observed only at 35%1RM, with higher values for the bench press exercise. It is worth noting that the results at different % loads during the incremental protocol for HF values analyzed in absolute units (data not shown) were the same as for HF values analyzed in normalized units, with lower values in arm exercise from 30%1RM compared to rest and also lower

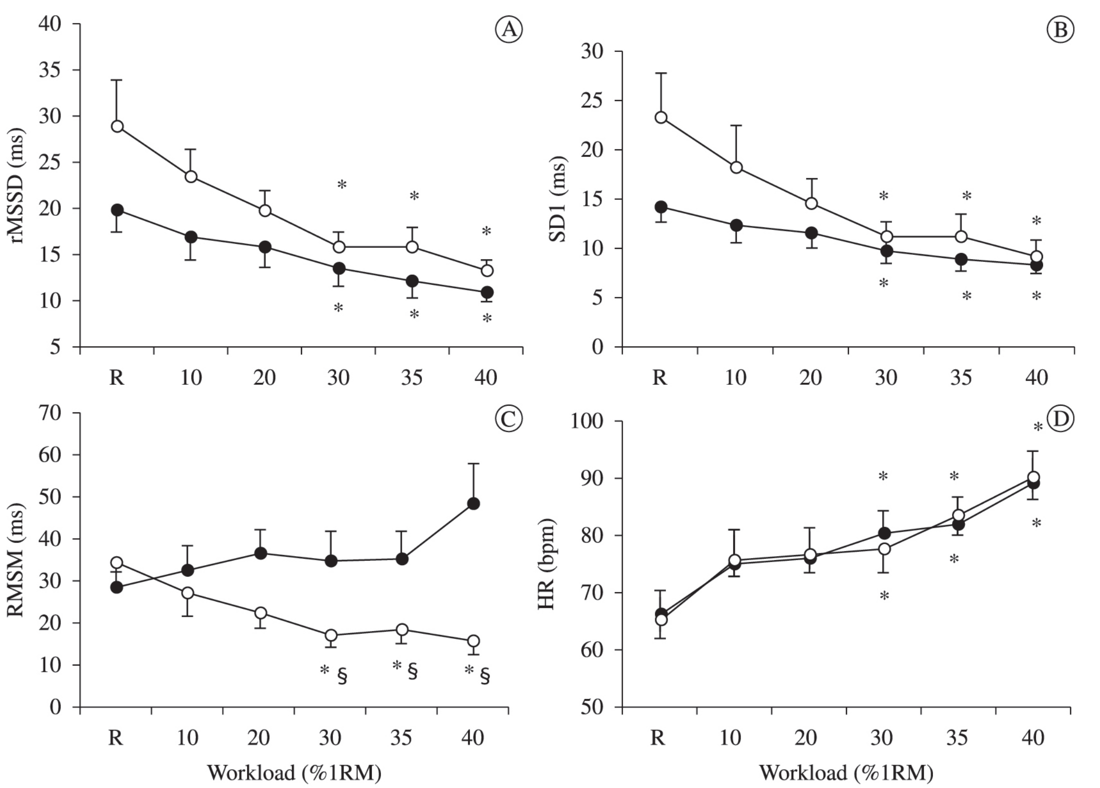

Figure 1. Heart rate variability indices at different loads of discontinuous resistance exercise protocol. (—●—) upper limb exercise; (—○—) lower limb exercise. Heart rate variability indices (A) rMSSD, (B) SD1, (C) RMSM, and (D) heart rate during incremental protocol. Data are presented as mean±SEM. R=rest; HR=heart rate; *p<0.05 compared to rest; § p<0.05 between exercises.

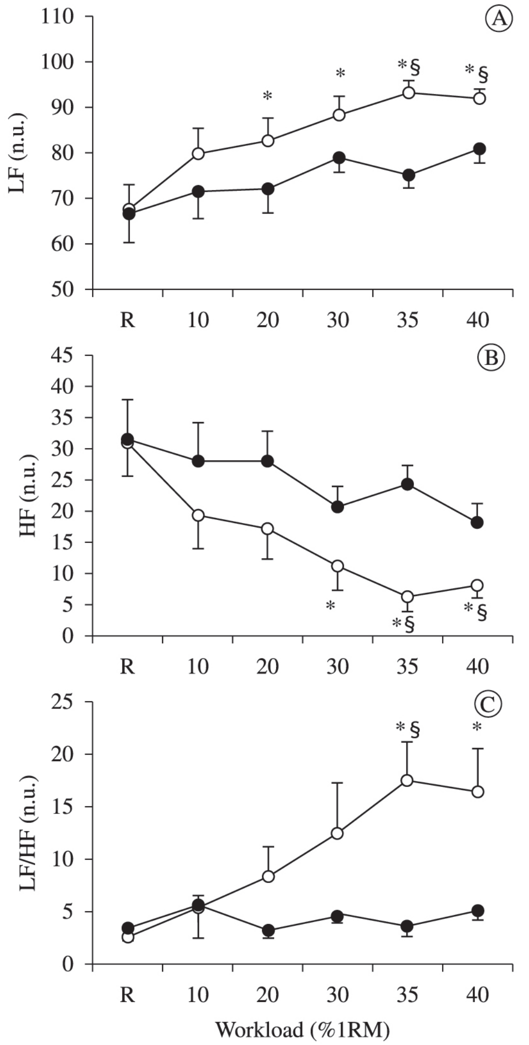

Figure 2. Frequency-domain indices of heart rate variability. (—●—) upper limb exercise; (—○—) lower limb exercise. (A) LF=low frequency; (B) HF=high frequency and (C) LF/HF; n.u.=normalized units. Data are presented as mean±SEM. *$p<0.05$ compared to rest; § $p<0.05$ between exercises.

values in bench press compared to leg press exercise at 35% and 40% of 1RM.

Discussion

The present study investigated the autonomic cardiac responses during upper versus lower limb resistance exercise at different loads in healthy older men. The main findings of this study were: 1) a significant reduction in the rMSSD and SD1 indices, representative of parasympathetic modulation, from 30% of 1RM in both exercises, but more pronounced in upper limb exercise; 2) a significant decrease in the RMSM index, representative of sympathovagal balance, from 30% of 1RM in the 45° leg press, and in contrast, an increase in the incline bench press; 3) a significant increase in HR from 30% of 1RM in both exercises.

To our knowledge this is the first study to evaluate and compare HRV (rMSSD, RMSM, and SD1) in acute upper and lower limb resistance exercise on the bench press and leg press at 45°. Several studies[2,17,21,29-31] evaluated HRV in leg resistance exercise but none compared the HRV response during a discontinuous incremental exercise in upper and lower limbs. Our results showed that, despite the different percentages of load applied, there was a predominance of sympathetic modulation in the upper limb resistance exercises. These results may contribute to understanding cardiovascular responses during this specific type of exercise in older people.

In the cardiovascular system, the behavior of cardiac function is mediated by neuro-regulatory activity from the autonomic nervous system. The electrical stimuli reaching the heart through these reflexive pathways are responsible for beat-to-beat variations and reflect sympathetic and parasympathetic modulation[27]. At rest, the parasympathetic modulation remains predominant, leading to lower values of HR and BP. However, during exercise or stressor stimuli, the sympathetic modulation on the HR becomes predominant and consequently leads to an increase in HR and BP[32].

In our study, the rMSSD and SD1 indices decreased as load increased. These indices reflect the vagal modulation[18,27,33] therefore, from 30% of 1RM, we can assume that there is a decrease in parasympathetic activity. However, it is not possible to determine the participation of the sympathetic branch by evaluating only these indices.

In this context, the RMSM index reflects both the sympathetic and parasympathetic modulation of HR[27,33]. In our study, the RMSM on the leg press presented lower values compared to the bench press from 30%1RM. In the inclined bench press, this index was stable with a slight increase throughout the load progression, while on the leg press there was a significant decrease in this index from 30%1RM.

From this result, we noted that total HRV differentiates between the two types of exercise, however, with this index it is not possible to reach conclusions about the autonomic branches separately. Although sympathetic and parasympathetic autonomic responses are not always reciprocal, in both exercises we observed a decrease in parasympathetic indices from 30% of 1RM without difference between them. So, we can only suggest a possibly higher sympathetic modulation in upper limb exercise compared with lower limb exercise.

At the same time, autonomic changes have been related to lactate accumulation at higher intensities, which can modify the autonomic modulation toward sympathetic predominance[21]. Simões et al.[21] showed that 30% of 1RM corresponded to the point at which aerobic metabolism is supplemented by anaerobic metabolism related to lactate accumulation in leg press exercise. This study, similarly to ours, also showed a decrease in SD1 and rMSSD from the same %RM, however, we did not evaluate lactate concentration to allow consistent inferences. Another study[34] involving an incremental exercise test on a bicycle ergometer demonstrated that all measures of vagal modulation decrease progressively until the ventilatory threshold level is reached, when sympathetic modulation begins to predominate.

In a previous study[35], there was a prevalence of sympathetic modulation on the HR after the anaerobic threshold point in dynamic exercises. The same behavior was found in the inclined bench press in the present study. However, even without a significant difference between exercises, the values for the arm exercise were lower than the leg exercise, suggesting stronger response in the arm compared to the leg exercise[17]. In a review, Helge[36] suggested that in arm exercise there is greater release of blood lactate compared to leg exercise, leading to a greater response of cardiovascular variables.

HRV spectral analysis was included to reinforce the results in the time domain. Similarly to previous studies that analyzed the frequency domain in short time series (2 and 3 min) we used 2 min segments during exercise[37-40]. Additionally, for HRV comparative analysis (rest and exercise), different time series were applied (5 min rest and 2 min of exercise), which has been shown previously[38].

In the frequency domain, we observed a decrease in HF and an increase in LF and LF/HF in the bench press when comparing rest and exercise. Some studies suggested that the HF spectral component is a marker of vagal modulation[41], LF is a marker of predominance of sympathetic modulation[42], and LF/HF may suggest sympathovagal balance[43]. Thus, our results suggest that, in the incline bench press from 30% 1RM, there was a shift toward sympathetic modulation.

The segments selected were 5 minutes of rest and visually the most stable exercise section containing 2 min free of noise at each load effort, excluding the initial 40 seconds of the exercise (period during which a rapid withdrawal of vagal activity occurred) and the final 30 seconds to avoid influences of blood pressure measurement.

In a previous study comparing arm and leg exercise, Tulppo et al.[18] verified faster vagal withdrawal during arm compared to leg incremental dynamic exercise performed on a cycle ergometer. The explanation can be the more powerful central command by vagal withdrawal and a stronger muscle afferent input during arm exercise compared to leg exercise. In our study, the vagal indices in the time domain did not present significant differences between exercises, although rMSSD and SD1 values were lower in the upper limb exercise than in the lower. However the HF index (in nu and ms^2) was also lower in the upper limb exercise compared to the lower limb exercise.

Considering the maximal load exercise, some authors[18] observed a higher level of sympathetic nervous activity during maximal lower limb exercise compared to upper, contrasting with our results. They support the idea of higher mass discharge of the sympathetic nervous system during lower rather than upper body exercise. These contradictory results may be explained by the type of protocol chosen. In our study, RE was performed discontinuously on resistance equipment (bench press and leg press) with different loads of 1RM, while the aforementioned study used continuous incremental exercise until exhaustion on a bicycle ergometer for legs and arms. Thus, the stimulus generated by the two types of protocols was different and can result in a distinct response.

Some differences in arm and leg exercise should be taken into account to better understand the results of the present study. In upper limb exercise, there is greater input possibly caused by the higher number and/or sensitivity of afferent receptors in this musculature[44]. Central command activation during RE depends on the exercise intensity and, consequently, the muscle tension, especially on the concentric phase of contraction, which can mechanically compress the peripheral arterial system causing an obstruction of blood flow. The mechanical and metabolic changes caused by exercise and muscle contraction can stimulate afferent fibers in type III and IV musculatures[32], with greater intensity in arm musculature. The metabolic changes in active musculature combined with reduced blood flow can stimulate mainly the type IV fibers which send information to the central nervous system, triggering an increase in sympathetic discharge to the cardiovascular system[45]. In arm exercise, the relative obstructed area is greater than in leg exercise and this is another aspect that may explain the higher sympathetic modulation in upper limb exercise.

The present study has certain limitations that need to be addressed, the first being the progressive exercise protocol design. Although it was a discontinuous incremental protocol, all loads were assessed in a single session, with an interval of 15 min between sets. This may have generated blood lactate accumulation and increased circulating catecholamine, thus influencing the HR responses mainly at higher loads and inducing volunteers to reach the peak load at a relatively low percentage of 1RM. Secondly, the RE protocol had a relatively long duration, however this was necessary to obtain a viable record for analysis. Lastly, the visual assessment of stationarity in the short-term HR recordings may be unreliable and can affect primarily the frequency-domain indices of HRV.

In summary, we can observe that significant cardiac autonomic changes occur from 30% of 1RM regardless of RE limb. However, there was more pronounced sympathetic increase and vagal decrease for upper limb exercises than for lower limb exercises. Therefore, this study provides a physiological understanding of HRV analysis comparing upper and lower limb exercise that can assist the physical therapist in prescribing RE more effectively to promote health in the elderly. Additionally, knowledge of autonomic responses in healthy older adults can serve as a benchmark for future studies involving the elderly population affected by chronic diseases such as heart disease.

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References

1. Pollock ML, Franklin BA, Balady GJ, Chaitman BL, Fleg JL, Fletcher B, et al. AHA Science Advisory. 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. http://dx.doi.org/10.1161/01.CIR.101.7.828
2. Williams MA, Haskell WL, Ades PA, Amsterdam EA, Bittner V, Franklin BA, et al. Resistance exercise in individuals with and without cardiovascular disease: 2007 update: a scientific statement from the American Heart Association Council on Clinical Cardiology and Council on Nutrition, Physical Activity, and Metabolism. Circulation. 2007;116:572-84. http://dx.doi.org/10.1161/CIRCULATIONAHA.107.185214
3. Aagaard P, Suetta C, Caserotti P, Magnusson SP, Kjaer M. Role of the nervous system in sarcopenia and muscle atrophy with aging: strength training as a countermeasure. Scand J Med Sci Sports. 2010;20:49-64. http://dx.doi.org/10.1111/j.1600-0838.2009.01084.x
4. Mayer F, Scharhag-Rosenberger F, Carlsohn A, Cassel M, Müller S, Scharhag J. The intensity and effects of strength training in the elderly. Dtsch Arztebl Int. 2011;108(21):359-64. PMid:21691559 PMCid:PMC3117172. http://dx.doi.org/10.3238/arztebl.2011.0359
5. Cruz-Jentoft AJ, Baeyens JP, Bauer JM, Boirie Y, Cederholm T, Landi F, et al. Sarcopenia: European consensus on definition and diagnosis: report of the European Working Group on Sarcopenia in Older People. Age Ageing. 2010;39:412-23. http://dx.doi.org/10.1093/ageing/afq034
6. Fried LP, Hadley EC, Walston JD, Newman AB, Guralnik JM, Studenski S, et al. From bedside to bench: research agenda for frailty. Sci Aging Knowledge Environ. 2005;2005(31):pe24 http://dx.doi.org/10.1126/sageke.2005.31.pe24
7. Chodzko-Zajko WJ, Proctor DN, Fiatarone Singh MA, Minson CT, Nigg CR, Salem GJ, et al. American College of Sports Medicine position stand. Exercise and physical activity for older adults. Med Sci Sports Exerc. 2009;41(7):1510-30. http://dx.doi.org/10.1249/MSS.0b013e3181a0c95c
8. Forte R, De Vito G, Figura F. Effects of dynamic resistance training on heart rate variability in healthy older women. Eur J Appl Physiol. 2003;89(1):85-9. http://dx.doi.org/10.1007/s00421-002-0775-1
9. Hu M, Finni T, Zou L, Perhonen M, Sedliak M, Alen M, et al. Effects of strength training on work capacity and parasympathetic heart rate modulation during exercise in physically inactive men. Int J Sports Med. 2009;30(10):719-24. http://dx.doi.org/10.1055/s-0029-1225329
10. Ades PA, Savage PD, Cress ME, Brochu M, Lee NM, Poehlman ET. Resistance training on physical performance in disabled older female cardiac patients. Med Sci Sports Exerc. 2003;35(8):1265-70. http://dx.doi.org/10.1249/01.MSS.0000079044.21828.0E
11. Deschenes MR, Kraemer WJ. Performance and physiologic adaptations to resistance training. Am J Phys Med Rehabil. 2002;81(11 Suppl):S3-16. http://dx.doi.org/10.1097/00002060-200211001-00003
12. Banz WJ, Maher MA, Thompson WG, Bassett DR, Moore W, Ashraf M, et al. Effects of resistance versus aerobic

training on coronary artery disease risk factors. Exp Biol Med (Maywood). 2003;228(4):434-40. PMid:12671188.

13. Braith RW, Beck DT. Resistance exercise: training adaptations and developing a safe exercise prescription. Heart Fail Rev. 2008;13(1):69-79. http://dx.doi.org/10.1007/s10741-007-9055-9
14. Kang J, Chaloupka EC, Mastrangelo MA, Angelucci J. Physiological responses to upper body exercise on an arm and a modified leg ergometer. Med Sci Sports Exerc. 1999;31(10):1453-9. http://dx.doi.org/10.1097/00005768-199910000-00015
15. Volianitis S, Secher NH. Arm blood flow and metabolism during arm and combined arm and leg exercise in humans. J Physiol. 2002;544(Pt 3):977-84. http://dx.doi.org/10.1113/jphysiol.2002.023556
16. Volianitis S, Yoshiga CC, Nissen P, Secher NH. Effect of fitness on arm vascular and metabolic responses to upper body exercise. Am J Physiol Heart Circ Physiol. 2004;286(5):H1736-41. http://dx.doi.org/10.1152/ajpheart.01001.2003
17. Okamoto T, Masuhara M, Ikuta K. Upper but not lower limb resistance training increases arterial stiffness in humans. Eur J Appl Physiol. 2009;107(2):127-34. http://dx.doi.org/10.1007/s00421-009-1110-x
18. Tulppo MP, Mäkikallio TH, Laukkanen RT, Huikuri HV. Differences in autonomic modulation of heart rate during arm and leg exercise. Clin Physiol. 1999;19(4):294-9. http://dx.doi.org/10.1046/j.1365-2281.1999.00180.x
19. Wilborn C, Greenwood M, Wyatt F, Bowden R, Grose D. The effects of exercise intensity and body position on cardiovascular variables during resistance exercise. Journal of Exercise Physiology Online. 2004;7(4):29-36.
20. Machado HG, Simoes RP, Mendes RG, Castello V, Di Thommazo L, Almeida LB, et al. Cardiac autonomic modulation during progressive upper limb exercise by patients with coronary artery disease. Braz J Med Biol Res. 2011;44(12):1276-84. http://dx.doi.org/10.1590/S0100-879X2011007500134
21. Simões RP, Mendes RG, Castello V, Machado HG, Almeida LB, Baldissera V, et al. Heart-rate variability and blood-lactate threshold interaction during progressive resistance exercise in healthy older men. J Strength Cond Res. 2010;24(5):1313-20. http://dx.doi.org/10.1519/JSC.0b013e3181d2c0fe
22. Kraemer WJ, Fry AC. Strength testing: development and evaluation of methodology: physiological assessment of human fitness champaign. Human Kinetics: 1995;115-138.
23. Borg G. Ratings of perceived exertion and heart rates during short-term cycle exercise and their use in a new cycling strength test. Int J Sports Med. 1982;3(3):153-8. http://dx.doi.org/10.1055/s-2008-1026080
24. Antila K. Quantitative characterization of heart rate during exercise. Scand J Clin Lab Invest Suppl. 1979;(153):3-68. PMid:293886.
25. Zuttin RS, Moreno MA, César MC, Martins LEB, Catai AM, Silva E. Evaluation of autonomic heart rate modulation among sedentary young men, in seated and supine postures. Rev Bras Fisioter. 2008;12(1):7-12. http://dx.doi.org/10.1590/S1413-35552008000100003
26. Mourot L, Bouhaddi M, Perrey S, Rouillon JD, Regnard J. Quantitative Poincaré plot analysis of heart rate variability: effect of endurance training. Eur J Appl Physiol. 2004;91(1):79-87. http://dx.doi.org/10.1007/s00421-003-0917-0
27. Heart rate variability. Standards of measurement, physiological interpretation, and clinical use. Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology. Eur Heart J. 1996;17(3):354-81. http://dx.doi.org/10.1093/oxfordjournals.eurheartj.a014868
28. Malliani A, Pagani M, Lombardi F, Cerutti S. Cardiovascular neural regulation explored in the frequency domain. Circulation. 1991;84(2):482-92. http://dx.doi.org/10.1161/01.CIR.84.2.482
29. Billman GE. Cardiac autonomic neural remodeling and susceptibility to sudden cardiac death: effect of endurance exercise training. Am J Physiol Heart Circ Physiol. 2009;297(4):H1171-93 http://dx.doi.org/10.1152/ajpheart.00534.2009
30. Motohiro M, Yuasa F, Hattori T, Sumimoto T, Takeuchi M, Kaida M, et al. Cardiovascular adaptations to exercise training after uncomplicated acute myocardial infarction. Am J Phys Med Rehabil. 2005;84(9):684-91. http://dx.doi.org/10.1097/01.phm.0000171167.31010.f4
31. Takahashi AC, Melo RC, Quitério RJ, Silva E, Catai AM. The effect of eccentric strength training on heart rate and on its variability during isometric exercise in healthy older men. Eur J Appl Physiol. 2009;105(2):315-23. http://dx.doi.org/10.1007/s00421-008-0905-5
32. Mitchell JH. J.B. Wolffe memorial lecture. Neural control of the circulation during exercise. Med Sci Sports Exerc. 1990;22(2):141-54. PMid:2192221. http://dx.doi.org/10.1113/expphysiol.2011.058156
33. Malliani A, Montano N. Heart rate variability as a clinical tool. Ital Heart J. 2002;3(8):439-45. PMid:12407819.
34. Tulppo MP, Makikallio TH, Takala TE, Seppanen T, Huikuri HV. Quantitative beat-to-beat analysis of heart rate dynamics during exercise. Am J Physiol. 1996;271(1 Pt 2):H244-52. PMid:8760181.
35. Sinski M, Lewandowski J, Abramczyk P, Narkiewicz K, Gaciong Z. Why study sympathetic nervous system? J Physiol Pharmacol. 2006;57 Suppl 11:79-92. PMid:17244940.
36. Helge JW. Arm and leg substrate utilization and muscle adaptation after prolonged low-intensity training. Acta Physiol (Oxf). 2010;199(4):519-28. http://dx.doi.org/10.1111/j.1748-1716.2010.02123.x
37. Vardas PE, Kochiadakis GE, Manios EG, Kanoupakis EM, Zouridakis EG, Chlouverakis GI. Spectral analysis of heart rate variability before and during episodes of nocturnal ischaemia in patients with extensive coronary artery disease. Eur Heart J. 1996;17(3):388-93. http://dx.doi.org/10.1093/oxfordjournals.eurheartj.a014870
38. Mendonca GV, Fernhall B, Heffernan KS, Pereira FD. Spectral methods of heart rate variability analysis during dynamic exercise. Clin Auton Res. 2009;19(4):237-45 http://dx.doi.org/10.1007/s10286-009-0018-1

39. Cottin F, Médigue C, Leprêtre PM, Papelier Y, Koralsztein JP, Billat V. Heart rate variability during exercise performed below and above ventilatory threshold. Med Sci Sports Exerc. 2004;36(4):594-600. http://dx.doi.org/10.1249/01.MSS.0000121982.14718.2A
40. Weippert M, Kumar M, Kreuzfeld S, Arndt D, Rieger A, Stoll R. Comparison of three mobile devices for measuring R-R intervals and heart rate variability: Polar S810i, Suunto t6 and an ambulatory ECG system. Eur J Appl Physiol. 2010;109(4):779-86. http://dx.doi.org/10.1007/s00421-010-1415-9
41. Malliani A. Principles of cardiovascular neural regulation in health and disease. Boston: Kluwer Academic Publishers; 2000. http://dx.doi.org/10.1007/978-1-4615-4383-1
42. Montano N, Porta A, Cogliati C, Costantino G, Tobaldini E, Casali KR, et al. Heart rate variability explored in the frequency domain: a tool to investigate the link between heart and behavior. Neurosci Biobehav Rev. 2009;33(2):71-80. http://dx.doi.org/10.1016/j.neubiorev.2008.07.006
43. Sandercock GR, Brodie DA. The use of heart rate variability measures to assess autonomic control during exercise. Scand J Med Sci Sports. 2006;16:302-13. http://dx.doi.org/10.1111/j.1600-0838.2006.00556.x
44. Ishida K, Takaishi T, Miyamura M. Ventilatory responses at the onset of passive movement and voluntary exercise with arms and legs. Acta Physiol Scand. 1994;151(3):343-52. http://dx.doi.org/10.1111/j.1748-1716.1994.tb09753.x
45. Iwamoto GA, Kaufman MP. Caudal ventrolateral medullary cells responsive to muscular contraction. J Appl Physiol. 1987;62(1):149-57. PMid:3558174.

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