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ORIGINAL ARTICLE

A shorter set reduces the loss of cardiac autonomic and baroreflex control after resistance exercise

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Abstract
Set configuration may affect the recovery pattern of cardiac vagal autonomic and reflex modulation after a resistance exercise, since it is closely associated with intensity and volume and determines the metabolic involvement of the session. We tested the hypothesis that longer set configurations have a higher impact on cardiac autonomic control and baroreflex sensitivity compared with shorter set configurations. We studied the effects of three set configurations with the same components of work on the cardiac autonomic control and baroreflex sensitivity. Seventeen subjects performed one control session and three experimental sessions of a leg-press exercise with the same volume (40 repetitions), resting time (720 s) and intensity (10RM load): (a) 5 sets of 8 repetitions with 3 min of rest between sets (8S), (b) 10 sets of 4 repetitions with 80 s of rest between sets (4S) and (c) 40 sets of 1 repetition with 18.5 s of rest between each repetition (1S). Longer set configurations (8S and 4S) induced greater reductions of the vagal cardiac autonomic control and baroreflex sensitivity ($p \leq .001$) compared with a shorter set configuration (1S). Also, 1S had non-significant reductions versus the control session ($p > .05$). These findings suggest that a shorter set configuration can reduce the impact of resistance exercise on the post-exercise cardiac vagal autonomic control and baroreflex sensitivity.

Keywords: *Cardiac autonomic control, baroreflex sensitivity, set configuration, resistance exercise*

Introduction

Long-term resistance training has been shown to be beneficial for prevention and improvement of musculoskeletal, metabolic or cardiovascular conditions (Hurley & Roth, 2000). In addition, resistance training improves several markers of cardiac autonomic control in both healthy (Piras, Persiani, Damiani, Perazzolo, & Raffi, 2014) and diseased individuals (Selig et al., 2004).

Heart rate variability (HRV) and heart rate complexity (HRC) are non-invasive methods to measure changes in autonomic modulation. HRV and HRC refer to the oscillation and irregularity of the cardiac cycles, respectively (Heffernan, Sosnoff, Jae, Gates, & Fernhall, 2008; Malik, 1996). A resistance-training session induced changes in HRV and HRC suggesting a transient reduction in cardiac vagal control after exercise (Kingsley & Figueroa, 2014). Also, a resistance exercise session may produce a decrement in baroreflex sensitivity (BRS) (Queiroz et al., 2013, 2014). Nevertheless, the effects of the loading parameters of resistance exercise on autonomic control and BRS are not fully understood (Kingsley & Figueroa, 2014).

In order to prescribe resistance exercise in a secure way, the effects of the loading parameters (i.e. intensity, volume, rest) on the cardiac autonomic and reflex control should be fully elucidated. Cardiac vagal control after a resistance session have been shown to be affected by intensity (Okuno, Pedro, Leicht, de Paula Ramos, & Nakamura, 2013) and volume (Figueiredo et al., 2015), meanwhile others have not confirmed these findings (Anunciação,

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Casonatto, & Polito, 2011; Kingsley et al., 2014). Another factor that could influence on the cardiac control is set configuration. Set configuration refers to the repetitions actually performed with regard to the maximum possible number of repetitions in a set. It is closely associated with intensity and volume, since it determines the total number of repetitions that can be performed prior to muscular failure (Iglesias-Soler, Carballeira, Sánchez-Otero, Mayo, & Fernández-del-Olmo, 2014) and modulates the metabolic involvement in the session (Iglesias-Soler et al., 2012).

Shorter set configurations, termed cluster training or inter-repetition rest training (Haff et al., 2003), result in a higher velocity and a lower glycolytic metabolism (Iglesias-Soler et al., 2012) than longer set configurations with repetitions close or leading to muscular failure. In addition, training protocols with shorter set configurations have revealed similar improvements in comparison with longer set configurations (Folland, Irish, Roberts, Tarr, & Jones, 2002). However, the cardiovascular responses to different set configurations have not been studied extensively. It is plausible that shorter set configurations may reduce the vagal withdrawal since strenuous protocols affect cardiac autonomic control (Okuno et al., 2013) and BRS (Niemelä et al., 2008) more than light protocols. A recent study comparing a resistance exercise protocol leading to muscular failure with another protocol with rests between repetitions did not find differences in the cardiac autonomic control (Iglesias-Soler, Boullosa, et al., 2014). However, the reduced volume used in that study and the high physical status of the participants may have prevented to induce sufficient fatigue in the participants to detect differences between protocols.

Therefore, the main goal of this study was to compare the effect of three resistance-training protocols equated in intensity, volume and work-to-rest ratio, but with different set configuration, on the recovery pattern of the cardiac autonomic control and BRS after exercise. In this sense, our aim is to identify the training protocol in which the heart control is less affected, which may have practical applications to prescribing resistance exercise in diseased individuals. Studies typically compare protocols differing in intensity, volume or rest. This impedes to know exactly which one is the variable that affects the cardiac control and to what extent to do. With our design, all these parameters (i.e. intensity, total volume, total rest and therefore the work-to-rest ratio) are strictly equated with except the repetitions performed in each set. Our hypothesis was that longer sets, with a lower velocity and hence a higher neuromuscular fatigue, will have greater impact on cardiac vagal autonomic and BRS recovery compared with shorter sets. If differences between protocols are due to set configuration, it is possible that shorter sets as an inter-repetition rest design may have practical applications to prescribe resistance exercise to diseased individuals in order to provoke a lower disturbance of the cardiac control after exercise.

Methods

Participants

Seventeen healthy adults (12 males and 5 females) participated in this study, with at least six months of experience lifting weights two or three times per week. Participants were screened and excluded if they had prior history of cardiovascular disease. The study was approved by the local Institutional Ethics Committee and participants signed an informed consent and were informed they could withdraw at any time. The characteristics of the participants are shown in Table I.

Procedures

A repeated measures design was used, in which participants completed a total of nine sessions: five orientation sessions and four experimental sessions. Participants were instructed to refrain from exercise, alcohol, caffeine and nicotine for 24 h and fast for 3 h prior to the testing sessions. Each session started with a warm-up of 5 min of submaximal cycling exercise and joint mobilization, and 2 sets of 10 repetitions using light loads.

Orientation sessions

Participants completed three familiarization sessions in which they were instructed on how to perform

Table I. Physical, cardiovascular and functional characteristics of the subjects ($n = 17$)

Characteristics	Values
Men/women	12/5
Age (year)	23 ± 2
Weight (kg)	68.6 ± 10.9
Height (m)	1.76 ± 8.6
Body mass index (kg/m^2)	21.8 ± 2.8
Resting HR (beats/min)	61 ± 14
Resting SBP (mmHg)	116 ± 9
Resting DBP (mmHg)	68 ± 7
Resting MAP (mmHg)	87 ± 7
10 RM in leg press (kg)	211 ± 45

Note: Data displayed as means ± SD.

the leg-press exercise programmes with a proper technique. Two sessions were performed subsequently, to test the 10RM and to establish reliability.

Dynamic leg press was performed using a diagonal sled-type double-leg-press machine (Biotech Fitness Solutions, Brazil). Participants were instructed to start with the knees fully extended and lowered until reaching a 90° of flexion of both knees and hip joints. After reaching this position, participants returned to the initial position performing each repetition as fast as possible. The same researcher provided verbal encouragement to the participants.

In order to obtain the 10RM load, a previously reported protocol was employed (Kraemer & Fry, 1995). 10RM was defined as the load that a participant was able to lift properly 10 times, but not 11.

Experimental sessions

Participants completed in an individual random sequence four experimental sessions, consisting of a control session and three exercise sessions with different set configurations. Participants did not know what protocols were going to be performed until the beginning of the session.

For each exercise session, the loading parameters (i.e. intensity, total volume and total rest) were equated in order to guarantee the same work-to-rest ratio.

Every exercise session consisted in a total of 40 repetitions and 720 s of rest, using the 10RM load. The exercise sessions differed according to the following set configurations: (a) 5 sets of 8 repetitions with 3 min of rest between sets (8S, with 8 repetitions performed over 10 possible repetitions [80%]); (b) 10 sets of 4 repetitions with 80 s of rest between sets (4S, 4 repetitions over 10 [40%]); (c) 40 sets of 1 repetition with 18.5 s of rest between each repetition (1S, 1 repetition over 10 [10%]). The control session (C) consisted of maintaining a semirecumbent position (i.e. exercise position) for 15 min. Sessions were separated by at least 72 h and were performed at the same time of the day (± 1 h) for each participant. A schematic representation of the experimental sessions is presented in Figure 1.

Physiological recording

A Task Force Monitor (TFM, CNSystems, Austria) was used for continuous monitoring of the heart rate (HR) and blood pressure (BP). HR was obtained by a three-lead electrocardiogram with a sampling frequency of 1000 Hz. Beat-by-beat monitoring of BP was obtained by photoplethysmography. Two pneumatic cuffs were placed on the proximal phalange of the index and the middle fingers of the left hand allowing a continuous BP measurement. The TFM has an additional oscillometric device that automatically and continuously transforms the absolute values of the finger pressure into the values of the brachial artery. The oscillometric device was located on the right arm. Data were obtained 10 min before and in the period 20–40 min after the end of the exercise. During this time, participants were seated, breathing spontaneously, in a semirecumbent position in the

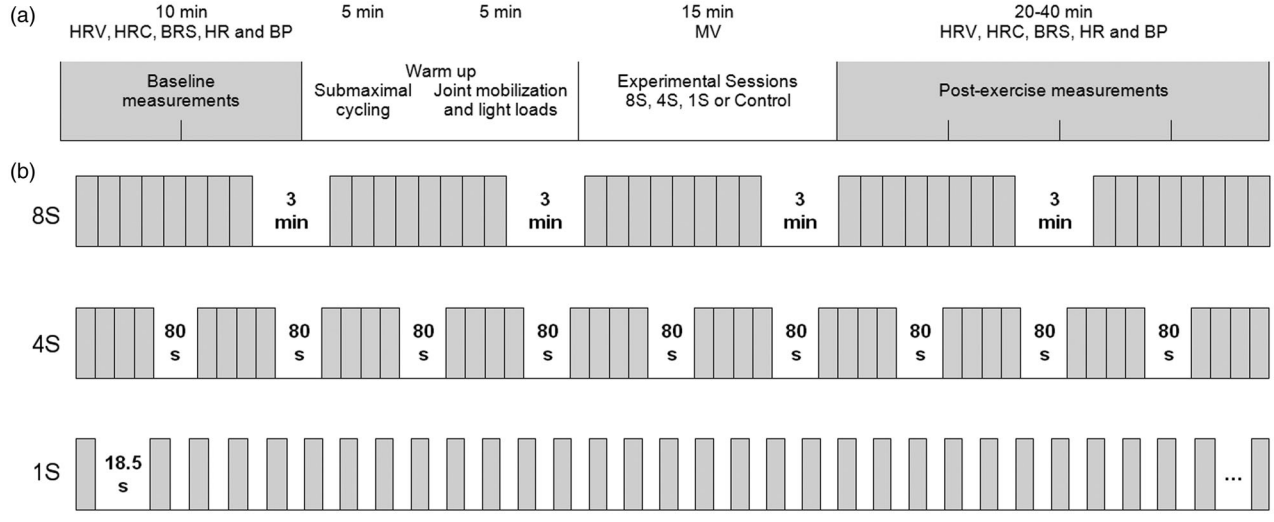

Figure 1. Schematic representation of the sessions. (a) Graphical simplification of the entire protocols design. HRV, heart rate variability; HRC, heart rate complexity; BRS, baroreflex sensitivity; HR, heart rate; BP, blood pressure; MV, mean velocity. (b) Representation of the experimental sessions. All sessions consisted of 40 repetitions and 720 s of total rest with the 10RM load. (8S) 5 sets of 8 repetitions with 3 min of rest between sets. (4S) 10 sets of 4 repetitions with 80 s of rest between sets. (1S) 40 sets of 1 repetition with 18.5 s of rest between each repetition.

leg-press machine. Data acquisition started after a period of 20 min post-exercise in order to avoid the effect of the increased respiratory rate on the autonomic parameters (Kingsley & Figueroa, 2014).

Physiological assessment

We analysed the HR, systolic blood pressure (SBP), diastolic blood pressure (DBP) and mean arterial pressure (MAP) over the last 5 min before the beginning of exercise. These variables were also evaluated for 5 min epochs across the 20–40 min obtained at the end of the protocols.

HRV was used to estimate the vagal autonomic modulation. Analysis of the data consists of time domain and frequency analyses. For the time domain analysis, the root mean square of differences between adjacent R–R intervals (RMSSD) was selected as an indicator of the vagal control of the heart (Malik, 1996). For the spectral analysis, Fast Fourier Transformation method with the Welch's method was employed (window width: 256 s, overlapping: 128 s). High-frequency activity (HF, 0.15–0.4 Hz) and low-frequency activity (LF, 0.04–0.15 Hz) in absolute units were calculated. HF is a marker of the cardiovagal control; meanwhile LF is mediated by both sympathetic and parasympathetic activities (Malik, 1996). To control the decreases in total power, normalized units of LF (LFnu) was used along with the LF/HF ratio, which are considered as markers of sympathovagal balance (Pagani et al., 1986). Epochs of 5 min were used as recommended by guidelines for HRV analysis during short-term recording (Malik, 1996).

Analysis of HRC was performed with Sample entropy (SampEn). While HRV determines the variability of the data, HRC determines the irregularity of these data. HRC measures are independent markers of parasympathetic modulation that yield essential information on HR dynamics (Kuusela, Jartti, Tahvanainen, & Kaila, 2002). SampEn is an indicator of system complexity that agreed more closely with the theory of random numbers than other entropies. SampEn determines the probability of finding specific patterns in a range from 0 to 2, being less predictive (i.e. complex) when values are close to 2 (Richman & Moorman, 2000). After the removal of lineal trends, an embedding dimension m (i.e. length of sequences to be compared) of 2 was used. The filter parameter r (i.e. tolerance for accepting matches) was set at 20% of the standard deviation (SD) of the time series and epochs of 5 min were used to the analysis following the suggestions published elsewhere (Heffernan et al., 2008).

Automatic artefact correction (i.e. medium correction threshold level, ± 0.25 s) and calculation were performed using Kubios HRV software v2.1 (The Biomedical Signal and Medical Imaging Analysis Group, UEF, Finland). Data were detrended with the smoothness priors method (Lambda: 500). Artefact correction never exceeded the 3% of the signal.

BRS was calculated using the sequence method (Bertinieri et al., 1988) with the TFM software v2.3. The sequence method consists of sequences formed by three or more consecutive beats of SBP and pulse intervals of their following beat (Lag 1), changed in the same direction. Thresholds were defined for 1 mmHg and 4 ms. Data analysis was performed for the last 10 min obtained before the protocol and for the intervals 20–30 and 30–40 min obtained after the protocols. Epochs of 10 min are usually used to analyse BRS after resistance exercise (Niemelä et al., 2008; Queiroz et al., 2014).

Velocity measurement

Velocity was recorded during exercise with a dynamic measure device (T-Force System, Ergotech, Spain). Mean velocity (MV) of the concentric phase of each repetition was calculated and averaged over the whole protocol of each experimental session (1S, 4S and 8S). MV was used as an indicator of neuromuscular fatigue, since the loss of velocity is related to metabolic production (Sánchez-Medina & González-Badillo, 2011). Neuromuscular fatigue is a reduction of performance as a consequence of a limited capability to generate force due to a neural or metabolic origin.

Statistical analysis

Descriptive statistics are shown as mean ± SD. Intraclass correlation coefficient (ICC) with single measure intra-class correlation was used to test the reliability of the 10RM (ICC = 0.989). A one-way repeated measures analysis of variance (ANOVA) was used to evaluate the effect of session (1S, 4S or 8S) on the averaged MV of every repetition. A two-way repeated measures ANOVA (session × time) was performed to evaluate the effect and interaction between session (1S, 4S, 8S or C) and time (Pre and 20–25, 25–30, 30–35, 35–40 min epochs for HR, BP and HRV markers; and Pre and 20–30, 30–40 min periods for BRS). Normality was tested using Shapiro–Wilk test. If data violated normality,

they were log transformed. *Post hoc* comparisons were performed with Bonferroni correction. A $p \leq .05$ was established as statistical significance. The data were analysed using SPSS 17.0 (SPSS, Inc., Chicago, IL, USA). A *post hoc* power analysis was calculated using the G Power software (version 3.1.4). Statistical power $(1 - \beta)$ of a repeated measures ANOVA with 3, 4 and 5 measurements for a sample size of 17, and a correlation among repeated measures of 0.5 and a medium effect size ($f = 0.25$) is 0.75, 0.64 and 0.71, respectively.

Results

Autonomic and baroreflex data

Autonomic and reflex data are shown in Table II. Values before exercise were similar between protocols for all variables ($p > .05$). For Ln of RMSSD, main effects for session ($F_{3,\ 48} = 5.491$ $p = .003$) and time ($F_{4,\ 64} = 7.732$ $p = .004$) were observed. The main effect of session revealed that 8S was significantly lower than Control session ($p = .01$). The main effect of time showed that the epoch of 20–25 min ($p = .028$) was significantly lower than the Pre values. Also, an interaction between session and time was observed ($F_{12,\ 192} = 13.580$, $p < .001$). 8S revealed lower values of RMSSD compared with the Control session and Pre values during the post-exercise period (20–40 min). Meanwhile lower values were observed for 4S in comparison with Control and Pre values for the 20–30 min interval. Also, RMSSD values were lower for 8S compared with 1S during 20–30 min period. No differences were found between 1S, the Control session or Pre values ($p > .05$).

For Ln of HF, main effects for session ($F_{3,\ 48} = 3.582$ $p = .02$) and time ($F_{4,\ 64} = 6.429$ $p = .004$) were observed. *Post hoc* pairwise comparison for the main effect of session did not reveal differences between protocols. The main effect of time showed that the epoch of 20–25 min ($p = .028$) was lower than the Pre values. Besides, an interaction between session and time was detected ($F_{12,\ 192} = 4.556$, $p = .003$). 8S had lower values of HF in comparison with the Control session and Pre values, for the 20–35 min period. In addition, lower values for 4S were observed in comparison with the Control session (20–30 min period) and Pre values (20–35 min period). No differences were observed between 1S and the Control session or between Pre recordings ($p > .05$).

SampEn showed a main effect of session ($F_{3,\ 48} = 5.115$, $p = .012$). Pairwise comparison revealed no differences among protocols. There was not interaction between session and time ($p > .05$).

For Ln of BRS, main effects for session ($F_{3,\ 45} = 4.756$ $p = .006$) and time ($F_{2,\ 30} = 15.385$ $p < .001$) were observed. The main effect for session revealed lower BRS values in 8S in comparison with Control session ($p = .007$). The main effect of time showed lower values of BRS for all the post-exercise period. The *p*-values of BRS for the periods were 20–30 min ($p = .002$) and 30–40 min ($p = .026$) with respect to baseline data. In addition to this, an interaction between session and time was observed ($F_{6,\ 90} = 5.902$, $p = .002$). 8S and 4S revealed lower BRS values compared with the Control session and the Pre values during the post-exercise period (20–40 min). Also, lower values were observed for 8S in comparison to 1S, for the 20–30 min interval. There were no differences between 1S and the Control session or Pre values.

For Ln of LF, a significant interaction between session and time was observed ($F_{12,\ 192} = 2.624$, $p = .016$). 4S had lower values of LF in comparison to the Control session in the 20–25 min period ($p = .038$) and in comparison to the 1S in the 25–30 min epoch ($p = .045$). No main effects were observed for this variable ($p > .05$).

For Ln of LF/HF, a main effect of time was observed ($F_{4,\ 64} = 7.932$, $p < .001$). The post-exercise epochs of 20–25 ($p = .006$), 30–35 ($p = .006$) and 35–40 min ($p = .026$) were higher than the Pre values. Neither the main effect for session nor the interaction between session and time were significant ($p > .05$).

LFnu showed a main effect of time ($F_{4,\ 64} = 8.105$, $p < .001$). Pairwise comparison showed higher values in the post-exercise periods of 20–25 ($p = .006$), 30–35 ($p = .008$) and 35–40 min ($p = .023$) in comparison to Pre values. There was not a main effect of session or an interaction between session and time ($p > .05$).

Haemodynamic data

Haemodynamic data are shown in Table III. For SBP, DBP and MAP, no main effects or interactions were observed among protocols ($p > .05$).

Velocity measurement

Values of MV were 0.29 ± 0.04 m s^{-1} for 1S, 0.27 ± 0.04 m s^{-1} for 4S and 0.26 ± 0.04 m s^{-1} for 8S. MV values showed a significant main effect for session ($F_{2,32} = 7.300$; $p = .006$). Pairwise differences were observed between 1S and 8S ($p = .016$) and between 4S and 8S ($p = .045$), with lower values for 8S.

Table II. Autonomic and baroreflex responses across sessions (n = 17)

	Pre	20–25	25–30	30–35	35–40
Ln RMSSD (ms)					
Control	3.97 ± 0.6	4.1 ± 0.49	4.14 ± 0.47	4.18 ± 0.47	4.16 ± 0.5
1S	4.01 ± 0.42	3.95 ± 0.53	3.96 ± 0.52	3.96 ± 0.53	3.95 ± 0.45
4S	4.1 ± 0.53	3.71 ± 0.54†‡*	3.77 ± 0.54†‡*	3.85 ± 0.5	3.89 ± 0.44
8S	4.18 ± 0.37	3.63 ± 0.59†*	3.71 ± 0.49†*	3.73 ± 0.55†*	3.79 ± 0.55†*
Ln HF (ms^2)					
Control	6.68 ± 1.38	6.93 ± 1.02	6.98 ± 0.96	6.99 ± 1.1	6.95 ± 1.2
1S	6.77 ± 0.78	6.38 ± 1.47	6.72 ± 0.96	6.63 ± 0.94	6.69 ± 0.82
4S	6.96 ± 1.08	6.14 ± 1.08†*	6.21 ± 1.14†*	6.3 ± 1.06*	6.56 ± 0.87
8S	7.11 ± 0.73	6.03 ± 1.17†*	6.21 ± 0.93†*	6.19 ± 1.06†*	6.37 ± 1.14
Ln LF (ms^2)					
Control	6.87 ± 1.09	7.51 ± 0.82	7.38 ± 0.89	7.41 ± 1.04	7.43 ± 1.11
1S	7.07 ± 0.86	7.18 ± 1.09	7.27 ± 0.82	7.24 ± 0.93	7.2 ± 0.88
4S	7.15 ± 0.92	6.77 ± 0.92†	6.75 ± 1.03‡	7.08 ± 1	7.15 ± 0.75
8S	7.24 ± 0.79	6.78 ± 1.24	7.01 ± 0.92	6.94 ± 0.85	7.12 ± 0.98
Ln LF/HF					
Control	0.19 ± 0.71	0.58 ± 0.78	0.40 ± 0.61	0.42 ± 0.84	0.48 ± 0.99
1S	0.3 ± 0.63	0.51 ± 0.68	0.55 ± 0.56	0.61 ± 0.81	0.51 ± 0.69
4S	0.19 ± 0.76	0.61 ± 0.68	0.54 ± 0.75	0.78 ± 0.66	0.59 ± 0.69
8S	0.13 ± 0.64	0.75 ± 0.85	0.8 ± 0.84	0.74 ± 0.81	0.75 ± 0.71
LF (nu)					
Control	54.07 ± 15.97	62.27 ± 17.38	59.01 ± 14.44	59.01 ± 18.81	60.31 ± 21.09
1S	57.13 ± 14.74	61.09 ± 15.4	62.48 ± 13.08	62.91 ± 17.17	61.31 ± 16.14
4S	54.28 ± 17.59	63.94 ± 14.95	62.03 ± 17.31	67.02 ± 14.4	63.08 ± 15.35
8S	52.72 ± 14.57	65.67 ± 18.47	66.88 ± 18.02	65.87 ± 17.83	66.25 ± 15.21
SampEn					
Control	1.8 ± 0.21	1.84 ± 0.16	1.82 ± 0.18	1.77 ± 0.25	1.83 ± 0.17
1S	1.77 ± 0.17	1.7 ± 0.21	1.76 ± 0.19	1.71 ± 0.17	1.72 ± 0.21
4S	1.69 ± 0.3	1.73 ± 0.29	1.78 ± 0.24	1.77 ± 0.25	1.68 ± 0.25
8S	1.76 ± 0.25	1.66 ± 0.37	1.65 ± 0.28	1.64 ± 0.25	1.61 ± 0.25
Ln BRS (ms/mmHg)					
Control	3.12 ± 0.58	3.12 ± 0.38		3.31 ± 0.47	
1S	3.05 ± 0.52	3 ± 0.43		3.04 ± 0.41	
4S	3.26 ± 0.53	2.8 ± 0.44†*		2.92 ± 0.39†*	
8S	3.21 ± 0.31	2.69 ± 0.49†‡*		2.8 ± 0.47†*	

Note: Data displayed as means ± SD.
†Significantly different versus C ($p < .05$).
‡Significantly different versus 1S ($p < .05$).
*Significantly different versus Pre ($p < .05$).

Table III. Haemodynamic responses across sessions (n = 17)

	Pre	20–25	25–30	30–35	35–40
SBP (mmHg)					
Control	114 ± 13	115 ± 9	113 ± 10	113 ± 10	113 ± 11
1S	115 ± 11	118 ± 12	119 ± 12	118 ± 11	118 ± 11
4S	117 ± 12	117 ± 16	117 ± 17	117 ± 17	116 ± 18
8S	117 ± 8	117 ± 11	117 ± 10	118 ± 9	116 ± 10
DBP (mmHg)					
Control	67 ± 11	71 ± 7	71 ± 7	71 ± 7	71 ± 8
1S	68 ± 8	70 ± 13	71 ± 14	71 ± 13	70 ± 14
4S	69 ± 9	71 ± 13	70 ± 14	69 ± 11	68 ± 11
8S	69 ± 7	73 ± 8	72 ± 8	72 ± 8	71 ± 9
MAP (mmHg)					
Control	85 ± 12	88 ± 7	88 ± 8	88 ± 7	88 ± 8
1S	86 ± 8	88 ± 13	89 ± 13	88 ± 13	88 ± 13
4S	88 ± 9	88 ± 13	88 ± 14	87 ± 12	87 ± 13
8S	87 ± 6	89 ± 8	88 ± 7	88 ± 7	87 ± 8

Note: Data displayed as means ± SD.

Discussion

The main finding of this study was that cardiac vagal autonomic control and BRS are affected by set configuration after a resistance exercise. The sets with higher number of repetitions (8 and 4 repetitions/set) induced the largest reductions of these parameters in comparison with the control session. Notably, no differences were observed between the shorter set configuration (1 repetition/set) and the control session.

Our results showed that when intensity, volume and work-to-rest ratio were equated, set configuration influenced the pattern of the cardiac vagal autonomic and BRS recovery. A plausible explanation for the differences between sessions may be attributed to the differences in glycolytic involvement between sessions, since vagal activity is negatively related with lactate production (Okuno et al., 2013; Simões et al., 2010). Although we did not assess lactate concentration, differences in velocity in our study and findings from previous studies support this explanation. On the one hand, differences in velocity strongly correlate with lactate production, with a higher lactate production in protocols with a lower velocity (Sánchez-Medina & González-Badillo, 2011). On the other hand, set configuration similar to our 1S is characterized by higher velocity and lower glycolytic involvement than a traditional configuration (Iglesias-Soler et al., 2012). Unfortunately, we did not measure lactate production and therefore we could not confirm this hypothesis.

The results of our study for longer set configuration (i.e. 8S and 4S) support previous findings showing that resistance exercise induce a reduction in cardiac vagal modulation (De Souza et al., 2013; Heffernan et al., 2008; Kingsley et al., 2014; Queiroz et al., 2013; Rezk, Marrache, Tinucci, Mion, & Forjaz, 2006). However, significant differences between resistance exercise protocols are scarce. Some studies have shown that intensity (Okuno et al., 2013) and volume (Figueiredo et al., 2015) may affect the vagal control of the heart, while others have not confirmed these findings (Anunciação et al., 2011; Kingsley et al., 2014). Contrary to our current data, a recent study (Iglesias-Soler, Boullosa, et al., 2014) comparing a resistance exercise protocol leading to muscular failure with another protocol with rests between repetitions did not find differences in the cardiac autonomic control. This discrepancy may be due to the reduced volume used in that study (~10 repetitions versus 40 repetitions in our study) or due to the differences in the fitness level of the participants (performance wrestlers versus an ordinary active population in our study), since it has been reported that volume (Figueiredo et al., 2015) and resistance-training experience (Kingsley et al., 2014) may influence the recovery of the vagal control after resistance exercise.

While the 8S and 4S configurations lead to a reduction of the cardiac vagal modulation, these changes were absent in the 1S configuration. 8S had a longer cardiac vagal withdrawal in comparison with 4S although 8S and 4S were not different in magnitude (i.e. size reduction in cardiovagal control). In addition, a single resistance exercise with longer set configurations was sufficient to reduce cardiac vagal control, as was previously reported in the literature (Iglesias-Soler, Boullosa, et al., 2014). Moreover, there were no differences between an inter-repetition rest design as the 1S and the control session (no exercise). This observation provides data that can be possible to perform resistance training without the cardiac impact that may imply a reduction in cardiovagal control to the participants (Albert et al., 2000).

Despite the differences between protocols due to the loss of variability, the analysis of complexity only revealed a main effect for session. As previously explained, complexity variables are independent markers of parasympathetic control that provide essential information of HR dynamics (Kuusela et al., 2002). In this sense, previous studies have reported reductions in complexity without changes in the variability parameters after a resistance session (Heffernan et al., 2008. Kingsley et al., 2014), suggesting that resistance exercise may affect more the complexity than the variability of the heart control. Contrary to this data, it seems that differences among diverse set configurations may be due more to a loss of variability in the signal than to a loss of complexity.

BRS was also affected by set configuration, with reductions for 8S and 4S, but not for 1S. Our results support previous findings showing that higher demanding protocols cause a decrease in post-exercise BRS (Niemelä et al., 2008; Queiroz et al., 2013). In our study, longer sets had a lower velocity than the shorter sets, and the loss of velocity indicates neuromuscular fatigue (Sánchez-Medina & González-Badillo, 2011). The reduced BRS observed in more strenuous set configurations may be due to a transient increase in arterial stiffness as a response to a higher sympathetic tone of the central arteries (Heffernan et al., 2008), since resistance exercise can affect the central vessels by producing a reduced wall deformation and hence an attenuated baroreceptor activation. Previous studies showed that a traditional resistance session with multiple exercises may affect BRS (Niemelä et al., 2008; Queiroz et al., 2013). In our study, we showed that a

single exercise with longer set configurations also affected BRS. Further, there were no differences between the 1S and the control session, which suggests that set configuration is useful in order to regulate the loss in the reflex control of the heart after resistance exercise.

These impacts in the cardiac control after exercise can be interpreted as a transient harmful effect in diseased individuals since 30 min after an exercise there is an increased possibility of a sudden cardiac death due to a decreased vagal activity (Albert et al., 2000). Also, prognosis studies revealed that reductions in both cardiac vagal modulation and BRS are associated with myocardial ischaemia and sudden cardiac death (La Rovere, Bigger, Marcus, Mortara, & Schwartz, 1998).

No post-exercise hypotension was observed after either protocol. The onset of post-exercise hypotension after resistance exercise is due to the interaction between the total volume performed, the muscle mass involved and reaching or not to muscular failure, in which the volume performed seems to be the main factor to provoke post-exercise hypotension (Figueiredo et al., 2015; Polito & Farinatti, 2009). Our results agree with previous studies in which similar protocols were insufficient to provoke changes in BP after resistance exercise (Polito & Farinatti, 2009).

Several limitations of the present study should be emphasized. Participants were healthy young adults; so the present findings should be taken with caution and further studies are needed in diseased individuals. We studied men and women in the same analysis, and genre may be a confounding factor. Also, glycolytic involvement was not measured. The inclusion of lactate production could provide further insight into the loss of cardiovagal autonomic control. As the cardiovascular parameters were not measured in the course of the exercise, it is not possible to know the physiological effects during the interventions. Finally, the breathing frequency and tidal volume were not controlled.

Conclusion

Our study suggests that a resistance-training session with a shorter set configuration design has a lower cardiovascular impact after exercise than longer set configurations, due to a lower disturbance of the autonomic and reflex control of the heart. The inter-repetition rest design did not induce a significant reduction in cardiac control. This finding may have practical applications in order to prescribe resistance training to diseased individuals. For instance, it could reduce the risk of a sudden cardiac death induced by a decreased vagal activity after an exercise. Further studies are needed in order to explore the effect of set configuration in the cardiac control of special populations. Also, a single resistance exercise may be sufficient to provoke a post-exercise reduction in cardiovagal autonomic control and BRS when is prescribed with longer set configurations. These findings provide evidence that cardiac vagal modulation and BRS are affected by set configuration after a resistance exercise, suggesting that set configuration could be a relevant factor to take into account when designing resistance exercise for diseased individuals. Future investigations should focus on identifying the effects on cardiac control of long-term resistance-training programmes differing in set configuration.

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Disclosure statement

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