Effect of the set configuration of resistance exercise on cardiovascular control and perceived exertion: Interaction with the type of exercise

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Abstract

After resistance exercise, there are reductions in cardiovagal control and baroreflex sensitivity, and decrements in blood pressure, that may have clinical relevance. This thesis present three studies that explored the interaction between set configuration and the type of exercise on the cardiac parasympathetic control, blood pressure, and perceived exertion. For this, maximal and submaximal sets were tested with exercises differing in the muscle mass involved. The results indicated that longer sets have a higher cardiac parasympathetic withdrawal and higher values of perceived exertion in comparison with shorter sets. Also, short sets with an inter-repetition rest design may not produce a reduction in cardiac parasympathetic activity. Submaximal sets did not affect post-exercise blood pressure but a long set leading to failure produced post-exercise hypotension in disregard of the exercise used. Lastly, the effect of set configurations on autonomic control and perceived exertion were dependant on the exercise performed, with dissimilar effects depending on the muscle mass involved and the set configuration used. This suggests that the prescription of resistance exercise through the set configuration may have important applications in training since it permits a control of cardiac parasympathetic reduction, the onset of post-exercise hypotension, and a modulation of perceived exertion.

Keywords: set configuration, muscle mass, resistance exercise, cardiac autonomic control, baroreflex sensitivity, post-exercise hypotension, perceived exertion

Resumen

Después de una sesión de fuerza, hay una reducción del control vagal cardíaco y de la sensibilidad barorrefleja, y una disminución de la presión arterial, que pueden tener relevancia clínica. Esta tesis presenta tres diferentes estudios en los que se exploran la interacción entre la configuración de la serie y el tipo de ejercicio en el control parasimpático cardíaco, la presión arterial y el esfuerzo percibido. Para esto, se testaron series máximas y submáximas con varios tipos de ejercicio con diferente masa muscular implicada. Los resultados indicaron que las series más largas producen una retirada parasimpática y un esfuerzo percibido mayor que las series más cortas. Además, las series cortas con un diseño de descanso entre repeticiones pueden no producir una reducción del control autónomo cardíaco. Las series submáximas no produjeron una reducción de la presión arterial pero una serie larga hasta el fallo muscular la produjo, independientemente del tipo de ejercicio realizado. Por último, el efecto de la configuración de la serie en el control autónomo y es esfuerzo percibido fue dependiente del tipo de ejercicio realizado, con efectos diferentes dependiendo de la masa muscular del ejercicio involucrado y de la configuración de la serie utilizada. Esto sugiere que la prescripción de fuerza a través de la configuración de la serie puede tener aplicaciones prácticas en el entrenamiento permitiendo controlar la reducción del control parasimpático cardíaco y la aparición de la hipotensión postejercicio, y modulando el esfuerzo percibido.

Palabras clave: configuración de la serie, masa muscular, fuerza, control autónomo cardíaco, sensibilidad barorrefleja, hipotensión postejercicio, esfuerzo percibido

Resumo

Despois dunha sesión de forza, hai unha redución do control vagal cardíaco e da sensibilidade barorreflexa, e unha disminución da presión arterial, que poden ter unha relevancia clínica. Esta tese presenta tres estudos no que se exploran a interacción entre a configuración da serie e o tipo de exercicio no control parasimpático cardíaco, a presión arterial e o esforzo percibido. Para isto, testáronse series máximas e submáximas con varios tipos de exercicio con diferente masa muscular implicada. Os resultados indicaron que as series máis longas producen unha retirada parasimpática e un esforzo percibido maior que as series máis curtas. Ademáis, as series curtas cun deseño de descanso entre repeticións poden non producir unha redución do control autónomo cardíaco. As series submáximas non produciron unha redución da presión arterial pero unha serie longa ata o fallo muscular a produxo, independentemente do tipo de exercicio realizado. Por último, o efecto da configuración da serie no control autónomo e o esforzo percibo foi dependente do tipo de exercicio realizado, con efectos diferentes dependendo da masa muscular do exercicio involucrado e da configuración da serie utilizada. Isto suxire que a prescripción de forza a través da configuración da serie pode ter aplicacións prácticas no adestramento, permitindo controlar a redución do control parasimpático cardíaco e a aparición da hipotensión postexercicio, e modulando o esforzo percibido.

Palabras chave: configuración da serie, masa muscular, forza, control autónomo cardíaco, sensibilidade barorreflexa, hipotensión postexercicio, esforzo percibido

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Men who have excessive faith in their theories or ideas are not only ill prepared for making discoveries; they also make very poor observations.
Of necessity, they observe with a preconceived idea, and when they devise an experiment, they can see, in its results, only a confirmation of their theory

— Claude Bernard, *An introduction to the study of Experimental Medicine*

Contents

Abbreviations

1RM	Maximum manifestation of strength
ANOVA	Analysis of variance
ATP	Adenosine triphosphate
BP	Blood pressure
BRS	Baroreflex sensitivity
DBP	Diastolic blood pressure
ECG	Electrocardiogram
H^+	Hydron
HF	High-frequency power
HR	Heart rate
HRC	Heart rate complexity
HRV	Heart rate variability
LF	Low-frequency power
LF/HF	Ratio between Low-frequency power and High-frequency power
Ln	Natural logarithm
MAP	Mean arterial pressure
MPV	Mean propulsive velocity of the concentric phase
MV	Mean velocity of the concentric phase
nu	Normalized units
PEH	Post-exercise hypotension
PCr	Phosphocreatine
RM	Maximum number of repetitions performed with a submaximal weight
RMSSD	Root mean square of differences between adjacent R-R intervals
SampEn	Sample Entropy
SBP	Systolic blood pressure
SD	Standard deviation
SEM	Standard error of the mean
$V0_2max$	Maximal oxygen consumption

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Study I

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1. General introduction

Resistance exercise has been shown to prevent and improve several musculoskeletal and metabolic conditions (1), and is recommended by the American College of Sports Medicine and the American Heart Association as a means to reduce pathologies related to these outcomes and improve the quality of life (2,3). In this sense, resistance training will lead to some health benefits, such as an increase of strength, muscle mass, and bone mineral density, and improvements in the metabolism of glucose, lipids, and lipoproteins (1–3). While the cardiovascular effects of aerobic exercise are studied traditionally, it was not until lately that the effects of resistance exercise on cardiovascular control were analyzed. In this regard, some efforts were made recently to design protocols to understand the effects of acute (4–7) and chronic (8–10) resistance exercise on the cardiovascular system and to improve the cardiovascular conditions of pathological individuals (8–10).

Nowadays, it is known that after a resistance training session, there is a reduction of cardiac parasympathetic control that is conducted by reductions in vagal autonomic activity (4,5,11–13) and glossopharyngeal and vagal baroreflex activities (5,14–16). That reduction in parasympathetic control may mean a transient harmful effect since 30 minutes after an exercise there is an increased risk of a cardiac event due to a decrease in parasympathetic activity in individuals at cardiac risk (17). Also, this reduction in parasympathetic activity should be managed in particular individuals such as athletes in order to prescribe exercise through the effect of the session on the nervous system for providing a better individualization of the training (18,19). Otherwise, it is known now that strength training produce acutely (20–22) and chronically (23,24) a reduction of blood pressure that could mean a reduction of the risk of suffer a coronary heart disease, stroke, or all-cause mortality (23). Nevertheless, while the beneficial effect of resistance training on blood pressure has been proven (23,25), the small number of studies performed until date has slowed the possible applicability of strength training as a non-pharmacological therapy to reduce or prevent high blood pressure (23,25).

In this sense, there is no consensus on how resistance training should be prescribed, due to the novelty of the field of study and the lack of applicable knowledge (2,26,27). There are multiple factors that could contribute to the effects of resistance exercise on cardiovascular system, such as loading parameters (i.e., load, volume, and suchlike), the type of exercise (i.e., upper vs. lower limbs), or the way to prescribe resistance exercise (i.e., percentage of 1RM, repetitions to failure, and suchlike). In this regard, the loading parameters, the type of exercises used, and the way to prescribe resistance exercise can modulate and determine the acute and chronic cardiovascular responses. Essentially, the former and the latter are interrelated since the way of prescribing resistance exercise may determine factors such as the load used, the repetitions performed, and the total rest of the session; and therefore the total volume performed or the ratio between work and rest. In summary, there is a lack of knowledge to indicate the precise responses of every variable of resistance exercise and how that contributes to undesirable or desirable effects on the post-exercise homeostasis, like reductions of cardiac autonomic and reflex control (15,28,29), or the onset of post-exercise hypotension (6,30,31) after resistance exercise.

Additionally, beyond the cardiovascular effects, resistance exercise provokes a perceptual response known as perceived exertion. Perceived exertion should be taken into account since control the effort during the work-out would offer the possibility to regulate the resistance exercise session and to know their physiological implication (32). The control of the process of strength training through perceived exertion with other indicators could prevent negative processes that lead to illness and overtraining (33), or determine the adherence to training (34). In this sense, despite the notion that some loading parameters determine and modulate the perceived exertion, such as the load (35), the total volume performed (36), or the rest periods between sets and exercises of the session (37), there is an absence of knowledge regarding how the effect of other parameters such as the set configuration or the type of exercise performed affect the perceived exertion.

Solving this issue is of importance in order to prescribe resistance exercise in a precise way, establishing correctly the loading parameters selected. Besides, knowing how the different loading parameters and the characteristics of the session affect the perceived exertion may help to understand in the future how the perceived exertion is determined by different mechanical, metabolic or neural processes. That may give clues about the possible physiological implication of the session and, therefore, signalize in the long-term the criterion-related validity of perceived exertion during resistance exercise.

Thus, the main goal of the present thesis is to analyze through three different studies how the way to prescribe resistance exercise by modifying the configuration of the set and the type of exercise selected, determine the cardiovascular responses, particularly the autonomic and baroreflex control and the post-exercise blood pressure, and the perceived exertion during exercise. It is hypothesized that long set configurations and an exercise with more muscle mass involved would produce a greater reduction in blood pressure and a larger withdrawal of cardiac autonomic and baroreflex parasympathetic control, concomitant with higher values of perceived exertion, in comparison with short set configurations and an exercise with less muscle mass involved.

1.1. How to prescribe resistance exercise?

To maximize the effect of resistance training, the exercise design may be manipulated by several variables, called loading parameters (38,39). Traditionally, the most important loading parameter is the intensity and refers to the load used (3). The importance of the intensity of load (40) lies in determining the total volume performed in a set prior to muscular failure (41).

1.1.1. Traditional prescription of resistance exercise

The intensity of load has been prescribed traditionally through two principal ways: the percentage of the maximum manifestation of strength (i.e. % 1RM) and the maximum number of repetitions that can be performed with a given submaximal weight (i.e., RM). Both mechanisms have advantages and disadvantages that should be known to maximize the effect of the prescribed resistance exercise program and to control the cardiovascular and perceptive (i.e., perceived exertion) effects that are provoked by the way of designate the workout.

Prescribing resistance exercise as a percentage of the 1RM is acknowledged as the most important stimulus related to changes in strength levels (42). Nevertheless, not all evidence supports that training with a certain percentage of 1RM is important for strength gains (43). It has the advantage that it can be used to program resistance training for many individuals at the same time while the loads can be easily transformed into absolute values (41). In this sense, prescription of exercise as a percentage of 1RM can clearly reflect the dynamics of the evolution of the training load (41). Nonetheless, the knowledge of the 1RM at a given intensity of load does not provide any accurate basis for prediction of how many repetitions can be performed at a given %1RM (44). This is because the number of repetitions that can be performed at each percentage of 1RM is exercise-dependent (45–50) and has a large inter-subject variability (45–50). During training, when resistance exercise is prescribed, a theoretical number of repetitions by set can mean that the muscular failure happens later or earlier than the repetitions prescribed. The former case may lead the individual to undertraining (51), while the latter leads to overtraining and injury due to the infeasibility of the repetitions determined (52).

An alternative worldview is to prescribe the intensity of load through the maximum number of repetitions that can be performed with a given submaximal weight. This approach associates an RM range with training goals, establishing a repetition maximum continuum (53). The continuum concept illustrates that a certain RM emphasizes a specific outcome, but, probably, the training benefits can be provided at any given RM (54). The RM approach has the advantage of eliminating the inter-subject variability and the differences between exercises (45–50). Nevertheless, the theoretical prescription of repetitions is correct for all individuals only in the first set of training. It is known that after the first set an individual may not be capable of performing the same number of repetitions, so this prescription is unrealistic (55,56) because the individual is not training within the prescribed RM. Otherwise, since the maximum number of repetitions that can be performed, at least in the first set, are well known, the possibility of undertraining is lower in comparison with the prescription through %1RM. Training to failure may have the advantage of a greater activation of motor units and a higher hormones secretion (52). Also, it is suggested that certain characteristics are better trained with ranges of RM than through the percentage of 1RM (57–59). However, performed over long periods, it has a high potential for overtraining and overuse injuries (52). Besides, training to muscular failure is not necessarily the best stimulus to improve strength gains (60).

1.1.2. An alternative prescription of resistance exercise: Set configuration

Another alternative prescription that could influence the acute responses and the strength gains after a training process is set configuration. Set configuration refers to the repetitions performed with regard to the maximum possible number of repetitions in a set (14–16). The importance of the relation between the number of repetitions performed and the total number of repetitions possible in a set has been analyzed from different perspectives and previously named as intensity (44,54,61,62), effort (40), intensity of effort (43) or level of effort

(41). In addition, for several authors, this relation between repetitions performed over repetitions possible is an independent loading parameter, as relevant as the intensity of load or volume (40,41,54,62). In this respect, several authors recommend prescribing resistance exercises considering the maximum number of repetitions (47,51,57,58,63), inasmuch as prescribing resistance through the percentage of 1RM disregards the maximum number of repetitions that can be performed, has a large inter-subject variability and is exercise-dependent (45–50).

1.1.2.1. Characteristics of the prescription through set configuration

Strength training prescription through set configuration is usually termed cluster training, inter-repetition rest training or intra-set rest training (64,65). Differences in terms refer to the total number of repetitions performed in each set, this is the intensity of effort (40), but they are essentially the same. In this regard, the configuration of the sets can be manipulated in two principal ways, once the intensity of load and the total volume are chosen: The number of repetitions performed in each set and the rest between each set or group of sets.

The number of repetitions performed in each set will show how far (short set configuration) or close (long set configuration) the set is with regard to the muscular failure. For example, with the 10 repetitions maximum (10 RM) load, it could be performed at only 1 repetition, with 9 repetitions left undone. This indicates a 1(10) set configuration, with an intensity of effort of 10%. However, if 4 repetitions were performed, with 6 repetitions left undone, it would show a 4(10) set configuration, with an intensity of effort of 40%. These two set configurations are examples of short set configurations. The first, 1(10), is termed inter-repetition rest training since individual repetitions are perform. The second, 4(10), is termed cluster training or intra-set rest training since the repetitions are performed in clusters.

On the other hand, the longer set configurations are performed close or leading to muscular failure. For example, when 8 repetitions are performed and only 2 repetitions left undone, this indicates an 8(10) set configuration, therefore with an intensity of effort of 80%. Finally, in an RM set configuration, when all repetitions are performed and 0 are left undone, this is a 10(10) set configuration so that intensity of effort would be maximal, this is, 100%. Graphical representations of these examples of short and long set configurations are shown in Figure 1.

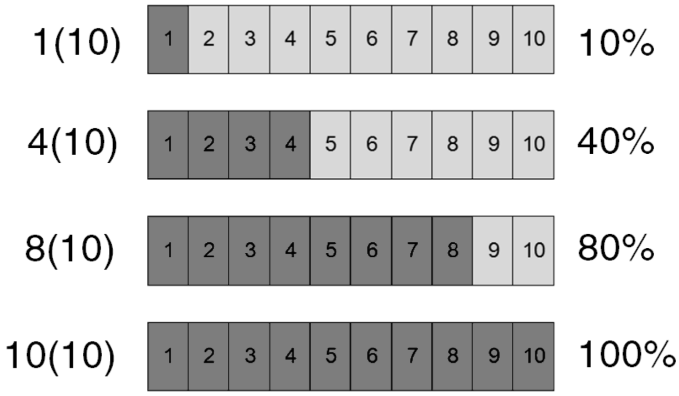

Figure 1. Representation of different examples of set configurations.

The rest between sets or group of sets are determined based on the metabolic replenishment needed to allow a more complete or incomplete recovery (64–66). In this regard, a break of about 15-30 seconds between repetitions or sets is typically left in short set configurations for an almost complete recovery (64,67), without additional benefits with longer rests (68). The effects of different set configurations will be discussed below.

1.1.2.2. Acute effects of prescription through set configuration

The different set configuration may lead to dissimilar acute 1) mechanical, 2) metabolic, 3) hormonal and 4) neural responses that should be taken into account to prescribe resistance exercise in a precise way.

1.1.2.2.1. Mechanical performance

Mechanical performance refers to the different expressions of strength in the assessment of resistance exercise movements (e.g., velocity, power or force). These expressions are important in resistance training because training to a higher mechanical performance, near or at optimal load (i.e., the load at which the greatest power output is observed), may produce favorable effects on training (69,70). In addition, as mechanical performance indicates the neuromuscular fatigue of the session (71), exercise to different mechanical performances can lead to dissimilar adaptations in a training process (70). In a set, after achieving the performance peak during the second or third repetition (72), there is a loss of performance with each new repetition (72,73). This reduction of mechanical performance achieves significant values when an individual performs between one third and half of the total number of repetitions, depending on the exercise performed (72,73). A short set configuration, with an intensity of effort lower than 50%, reveal a higher mechanical performance than long set configurations for several parameters including peak power (66,68,74–76), mean power (66,74), peak velocity (64,68,75), mean propulsive velocity (77), and peak force (68). Nevertheless, between short set configurations, there are not differences in such variables (68,75,78). This higher performance was observed for exercises of several modalities; in explosive barbell exercises such as clean pull (64) or power clean (68), in strength exercises such as bench press (78), leg press (66,74), or parallel squat (77), and in power exercises such as ballistic bench press (76), jump squat (75), or plyometric jump (79). The pattern of loss of mechanical performance in a long set configuration is always the same for a given exercise, independently of the load (73). Nevertheless, it is known that the loss of mechanical performance in short sets is less steep in comparison with long set designs (76). In this regard, previous studies have shown that the number of repetitions that can be performed until muscular failure with a short set is higher than with a long set design (80–82), up to 4-fold more (80).

1.1.2.2.2. Metabolic response

This differences in mechanical performance indicates the metabolic responses and, therefore, the neuromuscular fatigue of the session, as was previously pointed out (71). A resistance exercise induces different metabolic processes that occur simultaneously. There is a decrease in ATP stores and an increase in lactate and by-products of ATP (e.g., H^+) that contributes to fatigue (66,71). The extent of this reduction in ATP store and the increase in lactate depends on the configuration of the set (66).

Oxidative phosphorylation. The short set configurations maintain the PCr muscle content during exercise, meanwhile in long set configurations this content is reduced dramatically. This suggests that in short sets the ATP synthesis matched the ATP utilization, something that does not occur in the long sets (66). The extent of the degradation of PCr and the resynthesis of ATP is marked by the intensity of effort of the set and the rest between groups of repetitions or sets. Therefore, short set configurations have a lower disruption of the energy balance than long sets.

Anaerobic glycolysis. The changes in production of lactate, as a metabolic product of anaerobic glycolysis, occur simultaneously with the changes in PCr (66). Long set configurations have a higher glycolytic involvement than short sets (66,71,82–84). For short set configurations, a non-significant (83) or a slight elevation (66,82) of lactate is observed, suggesting that anaerobic glycolysis is extensively activated in long but not in short set configurations (66,71). This is because short sets may allow for a partial regeneration of PCr, resulting in low demand on anaerobic glycolysis and therefore less lactate production (66). These differences were observed between traditional, long set configurations versus inter-set rest (82–84) and inter-repetition rest (77) set designs.

When an insufficient rest is allowed between sets or repetitions (<15-30 s), no differences in lactate production are observed between short or long sets (81) due to a insufficient resynthesis of PCr in the short sets (64,67). It indicates that rest between repetitions or sets should be manipulated precisely to a reduced activation of anaerobic glycolysis.

1.1.2.2.3. Hormonal response

Previous studies have indicated that the metabolic involvement in a session determines in part the magnitude of the hormonal responses (85). As a consequence, enhancing the acute metabolic accumulation and hormonal response may result, in theory, in a higher cross-sectional area after resistance training (86). It seems that long sets of resistance exercise have a higher response of growth hormone (83,84), plasma epinephrine and norepinephrine (83), and cortisol (84) in comparison with short sets. Also, short sets produce a significant elevation of plasma norepinephrine (83), total and free testosterone (84) and cortisol (84) after a session, indicating a humble but significant hormonal response.

1.1.2.2.4. Neural response

Muscular activity increases with each new repetition performed, with the higher value near or at the end of the set, with the muscular failure (87). Due to this, the continuous pattern of the traditional set configuration results in a high threshold motor unit recruitment due to the size principle and, therefore, in greater muscular activity (88). In addition, higher threshold motor units are activated in response to an elevated metabolic production (i.e., lactate) that occurs in longer set configurations (89,90). Previous studies have observed a higher muscular activity in long set configurations versus short set configurations (81,91), confirming these postulations.

1.1.2.3. Chronic effects of prescription through set configuration

These acute differences in mechanical performance, metabolic involvement, hormonal and neural response between different set configuration sessions may lead to dissimilar adaptations to long-term training in strength, power, muscular endurance and cross-sectional areas.

1.1.2.3.1. Maximal strength

Most studies comparing the effects of training with different set configurations revealed no significant differences in maximal strength (70,83,92,93) or higher improvements in long set configurations (39,94,95). When higher maximal strength improvements were observed after long sets, short set configurations also improved significantly (39,94,95). Only one study reported higher improvements with shorter sets (69). The possible benefits for short sets may be due to the intensity of load used, since protocols using a medium-to-high intensity of load (<80%) had better o similar benefits (69,83,92,93) with short sets, meanwhile very high intensities of load (>80% 1RM) always provoked higher benefits for longer sets (39,94,95). Finally, comparables or different improvements between exercises seem to not be muscular or exercise-type dependent, since similar effects have been observed for the improvements of bench press (69,70,94), knee extension (83,92,93), and squat (39,69,70).

1.1.2.3.2. Power

Short set configurations seems advantageous for the improvement of power when training is performed near the optimal load (i.e., maximal power output) (69,70). Out of this point, benefits may be more humble (39), comparable (70,93), or lower (94) than with long sets.

However, contrary to the previous comment regarding maximal strength, improvements of power in training protocols differing in set configuration may be dependent on the muscles trained. The short sets seem to elicit a higher power development in comparison to long sets for lower limbs (e.g., squat) (39,69,70), but results for upper limbs (e.g., bench press) are at this moment confusing (69,70,94).

1.1.2.3.3. Muscular endurance

Improvements in the endurance with the same absolute load prior to training did not seem to be dependent on the set configuration used. In this regard, most of the cases reported revealed no changes between different protocols (70,83,93), with only one case reporting higher muscular endurance after training with long sets (70).

1.1.2.3.4. Cross-sectional area

Although a prior study revealed improvements in long set configuration (83), results are not conclusive, observing no differences in the majority of the cases (69,83). The theoretical model explains that the enhanced acute hormonal response in a series of sessions of strength training, as observed in long set configuration protocols, results chronically in an increased cross-sectional area (86). Nevertheless, this was not observed generally in studies that analyzed designs differing in set configuration (69,83).

1.2. Effect of resistance exercise on cardiac autonomic and baroreflex control

1.2.1. Cardiac autonomic control

The heart is not a metronome and the normal heart beat is not characterized by clockwork regularity. In healthy individuals maintaining normal sinus rhythm, changes in the heart period are expected (96). The modulation of the heart period is due to alternations in the autonomic activity originated by the cardiovascular centre in the medulla oblongata. The cardiovascular centre regulates the heart via changes in the activity of the parasympathetic and sympathetic fibres innervating the sinoatrial node. In this regard, parasympathetic or vagal activity slows the heart rate, meanwhile the sympathetic stimulation increase heart rate. Heart rate variability (HRV) and heart rate complexity (HRC) are non-invasive methods to measure these changes in the autonomic activity. The importance of these measures resides in that they are useful to monitoring aspects such as fatigue and recovery after training (18), and provide valuable information about the health and illness conditions dependent on the cardiovascular function of the individual (97).

HRV refer to the oscillation of the cardiac cycle (98) meanwhile HRC refer to their irregularity (12). This cardiac autonomic activity may be represented throughout different parameters with different time and frequency domain, and with non-linear methods (98). In this sense, the majority of the HRV parameters are surrogates of cardiac vagal activity between them (96). The HRC markers are also indicators of parasympathetic activity, but that yield essential information on heart rate dynamics (99).

1.2.1.1. Control of the cardiac autonomic activity after resistance exercise

After resistance exercise there is a reduction in the vagal control of the heart (4,5,11–13). The control of the reduction in vagal activity may serve to provide a better individualization of training prescription and to monitor aspects such as fatigue and recovery (18). Also, the control of the loss of vagal activity after resistance exercise may help to prevent possible outcomes such as sudden cardiac death in pathological individuals, since 30 min after an exercise there is an increased risk of an event due to a decrease in vagal activity (17). In addition, prognosis studies revealed associations between reductions in cardiac vagal control and myocardiac ischemia and cardiac death (97), justifying the importance of maintaining the maximal vagal activity possible after exercise.

It is well known that diseased individuals with cardiac antecedents have a dysfunctional autonomic control and therefore lower values of HRV and HRC in comparison with healthy individuals (100). Also, when there is a dysfunctional autonomic control, the recovery after exercise is worse in diseased individuals in comparison with healthy participants (100). Nevertheless, middle- and older-age hypertensive individuals with treated hypertension but with a intact cardiac autonomic control have the same cardiac nervous activity after resistance exercise than young- and middle-age healthy individuals (16,101). This makes young, healthy individuals an excellent model to try to understand how resistance exercise affects the cardiac control after a session.

Previous studies have reported that resistance exercise causes a greater autonomic disruption in comparison with endurance exercise (15,28,29). Thereby, one single exercise is enough to produce significant reductions in vagal control (102,103). Nevertheless, the cardiac impact of the different loading parameters of strength training remains still unknown. This impact must be elucidated in order to modulate the cardiac impact of the work out and prescribe exercise in an accurate and secure manner.

1.2.1.2. Effect of resistance exercise on cardiac autonomic control

As was explained before, studies reported a reduction in cardiac vagal control after resistance exercise in comparison with a control session (4,5,11–13); but attending to the loading parameters, only a few studies were published. It appears that the volume affects the cardiovagal control of the heart when the differences in total volume are massive (30), with a higher withdrawal when more total volume is performed. Nevertheless, it is not observed when the total volume and the differences in the total volume are minor (13).

Also, it seems that the intensity of load determines the cardiovagal withdrawal after resistance exercise. Most studies comparing the intensity of load revealed that medium or higher loads provoke a higher loss of parasympathetic control of the heart in comparison to lower loads (15,103–106), independent of the total volume being equated (103) or not (15,104–106). Nevertheless, the studies that were not equated (15,104–106) reached muscular failure at higher intensities. In this sense, reaching muscular failure may be a confounding factor that elicits *per se* a disturbance the autonomic control, beyond relative load.

In this regard, reaching to muscular failure provokes a high involvement of the glycolytic system and produces an important elevation of lactate on blood (71). Regarding the vagal cardiac control, it appears that the glycolytic involvement during resistance exercise determines the vagal withdrawal after the session (103,107). The relationship between the glycolytic involvement and the vagal withdrawal was observed previously at rest (108,109) and during aerobic exercise (110). On the one hand, it has been formerly pointed out that at rest there is a significant withdrawal of cardiac vagal control in the presence of lactate when it is injected intravenously (108,109). On the other hand, in the aerobic model of autonomic recovery after exercise, has been shown that the anaerobic involvement during the session modulates the cardiovagal withdrawal (110).

In resistance exercise, there is a lack of differences between protocols that are leading to muscular failure but that differ in volume and type of exercises (13), suggesting that reaching muscular failure provokes a huge reduction in the vagal control of the heart that is larger than the reduction that may be elicited by the loading parameters such as the intensity of load or the total volume performed (13). This has been previously shown in the aerobic model, in which the cardiovagal withdrawal was affected by the anaerobic involvement during the session while it was not affected by the total volume performed (110–112).

However, when different types of exercise (i.e., upper versus lower limbs) are compared between them with the same protocol but without reaching muscular failure, the difference between exercises exists, with a higher reduction of vagal control after the exercise of the lower limbs (113). This difference may be due to the dissimilar lactate production that exists between the exercises of upper and lower limbs (71).

At last, the cardiovagal response to resistance exercise in different resistance training status of the participants seems to be similar for HRV, despite that resistance trained individuals have a higher loss of HRC in comparison with moderately actively ones (13). This difference in HRC without changes in HRV is an interesting point of HRC, since as was argued before it yields essential information about heart rate dynamics beyond the surrogate markers of parasympathetic activity of HRV (99). Another previous study reported differences in HRC while it did not observe differences in HRV (12), suggesting that during some stressful situations or with some training status, the heart can experience a loss of complexity without a loss of variability (12,99).

1.2.2. Cardiac baroreflex control

The baroreflex is a mechanism that helps to maintain the homeostasis of the blood pressure. This mechanism responds to beat-to-beat changes in blood pressure by reflexively altering autonomic neural activity (114). The elevation of blood pressure results in a stretch of the aortic arch and the carotid sinus, where reside stretch-sensitive receptors. This deformation causes an increase in afferent neuronal firing at baroreceptive neurons. These afferent neuronal firing project to the dorsomedial medulla, which in turn signals the neurons composing the efferent autonomic limb of the baroreflex (115). The resultant cardiac adjustments modify the heart period to buffer the rises and falls of the blood pressure, preventing short-term wide blood pressure fluctuations, and thereafter reducing the variability of the blood pressure (116). A schematical representation of the baroreflex loop is shown in Figure 2.

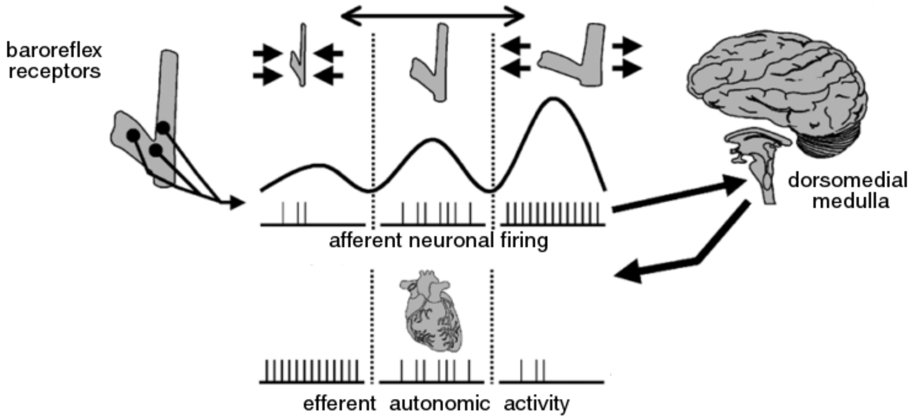

Figure 2. Schematical representation of the baroreflex loop. Adapted from Di Rienzo et al., (114).

The sensitivity -or gain- of the baroreflex (i.e., baroreflex sensitivity, BRS), is the relation between the changes in systolic blood pressure and their respective change in the heart period (117) around the operating point. The operating point is a non-fixed point where the systolic blood pressure and the heart are working under a given period of time previous to an invasive stimulus or during spontaneous changes (117).

Traditionally, this relationship is represented by a logistic model (i.e., carotid baroreflex function curve. Figure 3) where the changes in heart rate and blood pressure are analyzed in comparison with the changes in the carotid sinus pressure (118). These changes between blood pressure and heart period in the operating point, that is, the BRS, provide valuable information about health and illness conditions (119), is a predictor of cardiac mortality (97) and reveals valuable information about cardiac vagal autonomic activity (120).

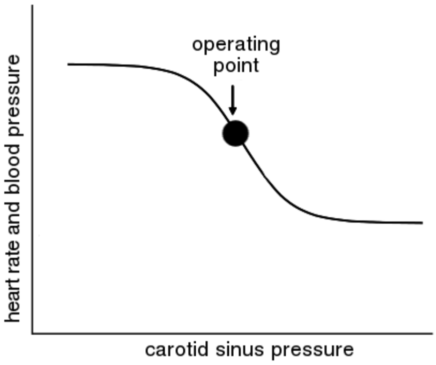

Figure 3. Model of carotid baroreflex function curve. Adapted from Raven et al., (118).

There are several techniques for the analysis of the BRS. This quantification has been measured traditionally by the injection of vasoactive drugs (i.e., Oxford technique). However, the need for intravenous cannulation and the use of drugs limit the applicability of this technique under real-life conditions (121,122). Recently, several techniques have been developed based on computer procedures that analyze the spontaneous changes between blood pressure and heart period and give precious information about the baroreflex control of the heart. These techniques, usually called “spontaneous methods” (123), identified by time domain (i.e., sequence method) (124) or frequency domain (i.e., α-index method) (125) the relationship between the changes of those variables.

From a general point of view, both methods report the same information about BRS (126). Nevertheless, the sequence method brings some advantages in comparison with the α-index method. These are: detailed information on the physiological minute-to minute variability of BRS, a reduced time window for the analysis, and offers a separate assessment of the reflex heart period induced by increases and decreases of heart period (122).

BRS by the sequence method is obtained by computing the slope of the regression line between three or more consecutive beats in which progressive increase or decrease in systolic blood pressure are followed by progressive changes of the heart period in the same direction (121). Also, BRS of the cardiac baroreflex control is predominantly parasympathetic, as observed by autonomic blockade in humans (127,128) and sino-aortic denervation in animals (124). Lastly, that the sequences reflect beat-by-beat interactions between blood pressure and heart period rather than chance coupling has been supported via surrogate data analysis (129).

1.2.2.1. Control of the cardiac baroreflex activity after resistance exercise

After resistance exercise there is a reduction in the BRS (5,14–16). In addition, after a resistance exercise session there is a great loss of BRS in comparison with an aerobic exercise session (14). While there is not a previously stated association between the reduction of the baroreflex control after exercise and an acute increased risk of a cardiac event; data interpretation suggest that the effect of resistance exercise on BRS should be monitored in order to avoid certain risks. In this sense, prognosis studies revealed that reductions in the level of BRS are associated with myocardiac ischemia and cardiac death (97), indicating the relevance of maintaining the maximal BRS possible after exercise.

Also, the decreased BRS could lead to a decrease in vagal activity, as the baroreflex function influences the resting cardiac vagal outflow (130). This could affect in the same way that exposed in the effects of resistance exercise on cardiac autonomic control section, since after resistance exercise there is a reduction of vagal activity (4,5,11–13).

Like in the case of cardiac autonomic control, it is known that diseased individuals with cardiac antecedents have a reduction in BRS in comparison with healthy individuals (119) and that when the participants have the same BRS between groups, the response of BRS to a resistance exercise session seems to be similar between groups (16). This makes healthy individuals a good model to analyze the effects of resistance exercise on baroreflex sensitivity in both health and disease when the baroreflex control is already working properly, in order to understand how the resistance exercise affects the baroreflex control.

In this sense, the effect of the different loading parameters of resistance exercise on post-exercise BRS is unknown yet. This impact must be elucidated in order to manage the cardiovascular impact of the work out and to prescribe resistance exercise in a precise and a safe way.

1.2.2.2. Effect of resistance exercise on cardiac baroreflex control

As was previously pointed out, it is known that after resistance exercise there is a reduction in the BRS, observed with the sequence (5,14,15) and α-index method (16). While a protocol with a higher intensity of load produced a longer reduction in BRS in comparison with a protocol with a lower intensity of load (15), the effects of other loading parameters are still unknown. Attending to this only previous study, could be postulated that more demanding protocols may cause a higher reduction of BRS, in comparison with less demanding protocols.

Thus, protocols with a higher intensity of load, more volume or a longer set configuration may produce a higher reduction of BRS. Several metabolites could account for this theoretically implication in more demanding protocols. In this sense, it was previously observed that while lactate infusion does not produce a reduction in BRS (109), the nitric oxide presence in blood produces a reduction in BRS (131). Nevertheless, no studies until date have analyzed these possible effects on BRS after resistance exercise.

1.2.3. Applicability of the set configuration on autonomic and reflex control

While most studies revealed no differences in the increase of maximal strength with different set configurations resistance training designs (70,83,92,93), the effect on cardiac autonomic and baroreflex control are probably dissimilar. As was previously commented, there is a reduction in cardiac vagal autonomic (4,5,11–13) and baroreflex control (5,14–16) after a resistance training session. The importance of controlling the cardiac autonomic and baroreflex loss lies in, on the one hand, the ability to provide a better individualization of training allowed by the prescription of exercise through the effect of the session on the nervous system (18); and, on the other hand, to help in diseased individuals to reduce the loss of cardiac control and, therefore, to prevent the possible outcomes associated with the reduction of vagal control, such as sudden cardiac death (17).

For this, to know the precise effects of the different loading parameters is strictly necessary. In this sense, the effect of set configuration is an interesting parameter to analyze, since permits to match the load, volume, and rest, and therefore the work-to-rest ratio. That allows to modulate the metabolic and perceptual responses while maintaining the loading variables equated. As was previously explained, glycolytic involvement during resistance exercise determines the cardiac vagal withdrawal after the session (103,107), so this is probably that the length of the set determines the cardiac autonomic control after resistance exercise. Additionally, the interaction with the type of exercise will be analyzed to better understand the effects of set configuration on cardiac control. In this sense, the type of exercise performed also determines the reduction in the cardiac control (113) due possibly to a different glycolytic involvement (71). It is hypothesized that longer set configurations would produce a higher cardiac vagal withdrawal in comparison with shorter set configurations and that this reduction would be increased with the use of type of exercises with muscle mass involved in comparison with exercises with less muscle mass involved.

In the case of baroreflex control, the factors that modulate the loss of BRS after resistance exercise are not known, but it is known that more demanding protocols cause a longer reduction of BRS in comparison with lighter protocols (15). In this regard, testing protocols equated in loading parameters but that allow for different intensities of effort can provide information about how this control is affected by the design of the session in general and about set configuration in particular. While the benefits of strength training are probably comparable regardless of set configuration used (70,83,92,93), knowing what designs help to better maintain the baroreflex control may have practical applications in diseased individuals. As in the instance of cardiac vagal control and due to its physiological relationship (132), since the baroreflex control of the heart is mainly parasympathetic (127,128), it is hypothesized that longer set configurations would produce a higher BRS reduction in comparison with shorter set configurations.

1.3. Effect of resistance exercise on post-exercise hypotension

1.3.1. Post-exercise hypotension

Post-exercise hypotension is a transient but sustained reduction in systolic and/or diastolic blood pressure below control levels after a bout of exercise. Post-exercise hypotension occurs in response to several types of exercise (133), but the purpose of this thesis focuses on post-exercise hypotension after resistance exercise. After resistance exercise, a maximal reduction of approximately 30/20 mmHg of systolic and diastolic blood pressure has been reported (31), lasting up to 24 hours in hypertensive individuals (134). Nevertheless, post-exercise hypotension is usually more humble, especially in normotensive individuals. Post-exercise hypotension after resistance exercise is commonly assumed to be less in magnitude and duration than after aerobic exercise (20), but when the type of exercises are compared between, this assumption is far of be demonstrated (135–138). While matching a session of aerobic training with a session of strength training is not possible since the units between types of training are different (i.e., ml/kg/min of $V0_2$ consumed versus Kg lifted), a acute session with the same relative load (i.e., % of the $V0_2$max versus % of the 1RM) produces comparable effects on post-exercise blood pressure (135,136).

The decrease in blood pressure observed after exercise could be an effective non-pharmacological strategy to treat or prevent the appearance of hypertension (139). In this sense, the acute reduction in blood pressure after exercise may contribute to chronic reductions in hypertensive individuals (140). In this sense, the magnitude of post-exercise hypotension correlates strongly with long-term blood pressure reductions produced by resistance exercise (141). Lastly, a meta-analysis suggested that resistance training may cause a significant reduction in systolic and diastolic blood pressure (23), so resistance training may be of clinical interest in the treatment or prevention of hypertension.

Studies that analyze the physiological process that lies in post-exercise hypotension are normally carried out with aerobic exercises and in a normotensive population. Also, studies that analyze the effect of the different loading parameters of exercise in post-exercise hypotension, for both aerobic and resistance exercises are normally performed in normotensive individuals. Thus, studies in resistance exercise performed in a hypertensive population are scarce (31,104,134,142–146). Nevertheless, it is possible that the differences in blood pressure after exercise between hypertensive and normotensive individuals are just quantitative and not qualitative in regard to the subjacent mechanisms of the post-exercise hypotension (16,133,147). Despite this, some authors suggest that these differences may be due to statistical phenomena as the regression toward the mean (148).

1.3.2. Physiological model of post-exercise hypotension in aerobic exercise

In the literature, there is just one general model based in aerobic studies to explain the physiological processes of the reduction in blood pressure after exercise. This model is defined in 1) obligatory mechanisms, that are essential to the onset of post-exercise hypotension, and 2) situational influences, that vary from one study to another (147).

1.3.2.1. Obligatory mechanisms

The obligatory mechanisms to the onset of post-exercise hypotension are a resetting of the baroreflex, mediated by a central decrease in sympathetic outflow (149–151), a blunted transduction of sympathetic outflow into vasoconstriction (149), and a sustained histaminergic vasodilatation (152,153).

After aerobic exercise, the baroreflex control is well preserved (154) but is reset in order to defend lower blood pressure (149,155). This resetting of the baroreflex results in reduced sympathetic outflow that is observed by a reduction of the muscle sympathetic nerve activity in both normotensive (149) and hypertensive (150,151) individuals. As muscle sympathetic nerve activity is reduced after exercise rather than elevated, as might be expected during a reduction of blood pressure (150), baroreflex-mediated regulation of the muscle sympathetic nerve activity must be reset to a lower operating point during postexercise hypotension (139).

In addition to this, postexercise hypotension is associated with a blunted transduction of sympathetic outflow into vasoconstriction (149). It means that for a comparable levels of muscle sympathetic nerve activity, there is an attenuated vascular resistance in the previously active muscles (149). An increased reuptake of noradrenaline or a presynaptic inhibition of noradrenaline release are possible explanations for this blunted transduction (147).

Also, there is a postexercise vasodilatation dependent on the activation of histamine H_1 (156) and H_2 (157) receptors. Combined H_1 and H_2 receptor antagonism reduce in a large extent the vasodilatation and hypotension that occurs after exercise (152,153). Histamine release can be elicited by a degranulation of the mast cells located within the connective tissue of the surrounding skeletal muscle, or by an increase in reactive oxygen species or a rise in temperature.

Also, histamine appears to be formed newly due to oxidative stress or as a consequence of shear stress in large vessels (147). Prostaglandins (158) and nitric oxide (159) appears to have little effect on the contribution of postexercise vasodilation.

1.3.2.2. Situational mechanisms

The situational mechanisms, contrary to the obligatory mechanism, vary from one study to another but affect the pathways that are related with post-exercise hypotension. These include the presence or absence of gravitational stress due to posture, and the fluid status and the heat balance with the environment. These mechanisms are manifested as changes in the stroke volume and hence in the cardiac output. The general model based in the majority of studies reports an elevated cardiac output during postexercise hypotension (160). This elevation is generally due to a well-maintained stroke volume and an elevation of heart rate. Also, hemodynamic mechanisms are dependent of the status of the participants (161).

The position in which the participants are tested after exercise influences the hemodynamic responses (162,163). Most of the studies reporting hemodynamic responses that have been carried out in a supine position indicate an elevation of cardiac output without changes in stroke volume. However, when studies are performed in a seated position, reports inform a possible reduction in cardiac output (161,162). This is due to a hindered venous return that affects cardiac preload and therefore stroke volume (162,164,165). Further, fluid loss or heat balance appears not to affect postexercise hypotension (166,167) though stroke volume appears to be highly affected by these factors (168). In this sense, heat stress leads to a fluid loss that decreases cardiac preload and hence decreases also stroke volume. On the contrary, the heat can lead to a increase in heart rate that augments cardiac output (168). The physiological model that explains the obligatory and situational mechanisms during post-exercise hypotension (147) is represented conceptually in Figure 4.

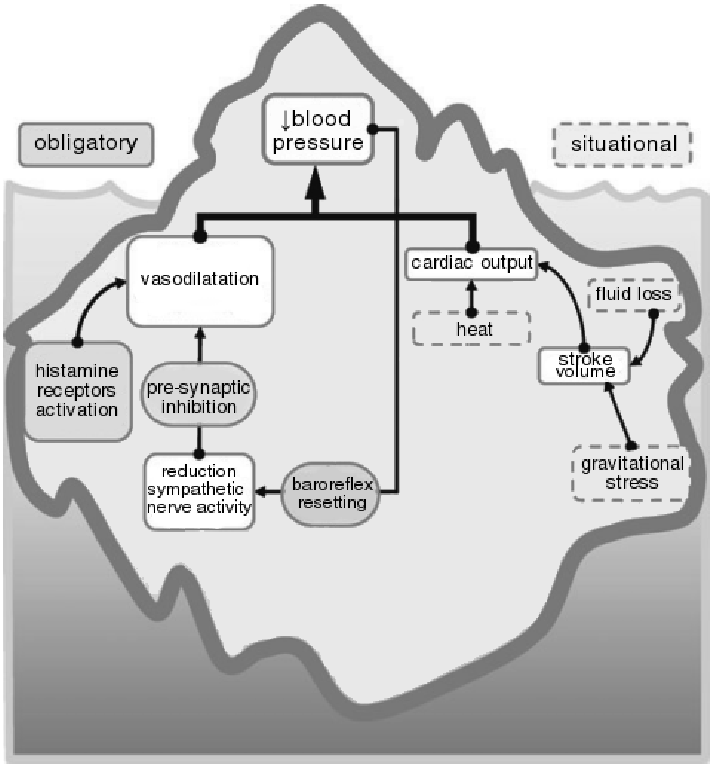

Figure 4. Obligatory and situational mechanisms during post-exercise hypotension. Based from Halliwill et al., (118).

1.3.3. Effect of resistance exercise on post-exercise hypotension

A resistance exercise session can cause an acute decline in the ambulatory blood pressure values in both normotensive (137,169,170) and hypertensive (134,143) individuals. In this sense, blood pressure may be declined after exercise during the periods of day (134,137,143), sleep (143,169,170) and during the 24 hours after a resistance exercise session (137,143,170). These reductions in ambulatory blood pressure have been observed for both systolic (134,137,143,170) and diastolic (134,137,143,169,170) blood pressure. In hypertensive individuals, there is always a reduction in systolic blood pressure after a resistance exercise session (31,104,134,142–145). These falls in the 24 hour period after a resistance exercise session can suppose a reduction of approximately 4 mmHg of systolic and/or 3 mmHg of diastolic blood pressure (137,143,170).

However, the main body of studies analyzes the effect of a resistance exercise session on post-exercise blood pressure between 60 and 90 minutes after the end of the session (30,106,171–174). Reductions are also observed for normotensive (16,30,106,173,175) and hypertensive (31,104,134,142,144) individuals, and for systolic (4,30,104,173,176) and diastolic (16,30,170,171,177) blood pressure. Although there are several studies in which post-exercise hypotension is not observed (6,178–183), when post-exercise hypotension is noticed, it normally lasts up to approximately 60 min with mean reductions of about 5-10 mmHg for systolic and diastolic blood pressure (16,30,106,171–173,184–186). However, in several studies the time under post-exercise hypotension matches the total time of data collection (30,106,171–174), so post-exercise hypotension may actually be longer.

Otherwise, the factors that affect postexercise hypotension after resistance exercise are not entirely understood. Thus, to make the prescription of resistance exercise useful to elicit post-exercise hypotension, it is necessary to understand the effects of the loading parameters in order to know the extent to which these parameters affect post-exercise blood pressure and how to maximize them. In this regard, the main differences between protocols are given by the time under post-exercise hypotension (i.e., duration) while differences in the size of the decrease of post-exercise hypotension (i.e., magnitude) protocols are scarce (4,30,31,104,106,176).

Although there are works about several parameters of the load, the main factors that might be responsible for the onset or increase of a hypotensive effect after resistance exercise are a) the total volume, b) type of exercise, c) intensity of load and d) the onset of muscular failure. It is important to note that these factors are interrelated and, therefore, they can affect each other.

1.3.3.1. Total volume

It appears that the total volume is the main factor to the onset of postexercise hypotension after resistance exercise. The more volume, the longer the duration of the hypotensive effect for both normotensive (30,172) and hypertensive (142) individuals. Besides, total volume seems to be the only load parameter by which the magnitude of reduction of blood pressure can be widely modulated (30). In this sense, normotensive individuals after performing a session with large volumes experience lower blood pressures than individuals after a session with small volumes for the entire postexercise period (30). Also, protocols with very low total volume do not produce a hypotensive effect (6,179–181,187,188).

The type of protocol (i.e., paired exercises protocols versus traditional designs) does not appear to be important for the onset of postexercise hypotension when the total volume is equated, and the same exercises are performed (172). Nevertheless, when differences in total volume are observed between protocols due to reaching muscular failure, the designs that have a higher volume produce a longer hypotensive effect (174,189,190). These protocols to muscular failure alternated agonist and antagonists exercises (190) or exercises of the upper and lower limbs (174,189) in comparison to designs performed in consecutive sets to muscular failure and that, therefore, allowed a lower total volume. All this also suggests a major role of volume on the hypotensive effect (174,189,190).

1.3.3.2. Type of exercise

It appears that the inclusion of exercises of the lower limbs (i.e., legs) help tremendously to induce the onset and duration of post-exercise hypotension, probably due to the muscle mass involved in the exercises (6,146,176). This is true even with small volumes performed (191,192).

When the protocols are performed with lower limbs of the body and leading to muscular failure, small total volumes in the session but with a great number of sets by exercise produces a hypotensive effect (6). Nevertheless, when the protocols are also performed leading to muscular failure but with the upper limbs (i.e., arms) no hypotensive effect is observed, independently of the number of sets by exercise (6). It seems that when there is a sufficient volume, the type of exercise is another important variable to provoke hypotensive effect.

In this sense, post-exercise hypotension may be longer in exercises of the lower limbs (176), or it can be absent after exercises in the upper limbs when it is observed in the lower limbs (6,146). Thus, there are several studies in which no effects were observed when the exercises were performed only in the upper limbs (6,146,172,188,193). Additionally, small volumes performed with the lower members have shown a hypotensive effect, with (191) or without (192) leading to muscular failure, an important co-factor of post-exercise hypotension (104).

1.3.3.3. Intensity of load

It seems that exercise at a medium intensity of load (60-70%) increase mildly the time under hypotension in comparison with high (80%) and low (40%) intensities of load for systolic (106) and diastolic blood pressure (106,171). Also, an intensity of load about 30% (176,194) or 40% (135,195) is enough to produce post-exercise hypotension, provided that a minimum of volume is made.

In hypertensive individuals, exercise at a high intensity of load (80%) showed a higher magnitude of reduction in comparison to low intensities of load (50%) for systolic and diastolic blood pressure during all the post-exercise period (31,104). Nevertheless, participants in the protocol of higher intensity of load reached to muscular failure (104), which can be a confounding factor.

In normotensive individuals, differences of magnitude were observed only in specific moments in the post-exercise period (i.e., not all the course of data collection). In these particular moments, protocols at high (80%) or medium (70%) intensities of load had lower values of systolic blood pressure in comparison with the counterpart with a relatively lower intensity of load (40% and 60%, respectively) (106,179). Nevertheless, the contrary was observed in other studies for systolic blood pressure (106) and diastolic blood pressure (4), with lower values in the relative lower intensities of load (40%, 60%) in comparison with higher intensities of load (70%, 80%). Also, protocols at lower intensities of load affected to diastolic blood pressure in several studies in which the protocols at high intensities of load did not affect (172,176,182,196). Taking all this into account, to prescribe resistance exercise at the medium intensity of load may be recommendable in order to descend the systolic blood pressure (106,179) while prescribing resistance exercise at the low intensity of load could be a better option when the objective is to lower the diastolic blood pressure (4).

1.3.3.4. The onset of muscular failure

Reaching muscular failure may be another co-factor that determines the onset and the increase of post-exercise hypotension and that has not been studied sufficiently (104). When resistance exercise is prescribed through RM and, therefore, muscular failure is intentional, post-exercise hypotension is observed even with low total volumes (6,142,190). Nevertheless, a minimum total volume may be necessary to the onset of post-exercise hypotension since protocols with very low total volumes do not produce this effect, even with the onset of muscular failure (6,15). Also, a protocol that led to muscular failure but that was larger in total volume produced a longer hypotensive effect than another protocol that also led to failure but that have a relatively lower volume (172), showing the importance of the volume performed beyond muscular failure itself.

Besides, when protocols are equated in total volume, the protocols that lead to muscular failure produce longer hypotensive effects than protocols that do not lead to muscular failure (11,172,196), indicating certain importance as a co-factor effect when the volume is equated. Unfortunately, the intensity of load between these protocols was different and thus may have affected the responses (106,171).

1.3.4. A physiological model for resistance exercise

As explained before, the physiological processes studies in post-exercise hypotension are normally performed in aerobic studies. So it is plausible that the factors that produce post-exercise hypotension differ in characteristics and magnitudes between aerobic and resistance exercise.

Some studies reported an increase in systolic blood pressure 1-5 min after the end of resistance exercise (135,178,182,197). So it is possible that the immediate post-exercise hyperemia that happens after aerobic exercise (198) and helps to provoke the first minutes of post-exercise hypotension does not occur to the same extent after resistance exercise.

Also, previous studies reported a decrease in cardiac output due to a reduction in stroke volume after resistance exercise (4,5,16,199), since heart rate is above (4,5,199) or not different (16) than control values. That is probably due to a decrease in the pre-load that occurs because of the change in fluids status from plasma to interstitial space during exercise (200,201) or due to cardiac fatigue as a consequence of loss of myocardial performance (165). After aerobic exercise, an elevation in cardiac output is usually reported (160), with exceptions depending on the status of the participants. That may be another difference between resistance and aerobic exercise models.

Moreover, an increase in systemic vascular resistance is commonly observed after resistance exercise (4,5,16) despite the fact that it was traditionally proposed in aerobic models that systemic vascular resistance might be lower after exercise (202). It is probably due to the decrease in cardiac output provoked by the shift in plasma volume, with individuals having an increased systemic vascular resistance that is not fully compensated due to the inhibition of the sympathetic vasoconstrictor nerves (147).

Differences reported in the literature in cardiac output and systemic vascular resistance between resistance and aerobic exercise models could also be affected by the gravitational stress of the posture (167,168). This is because the individuals in the resistance exercise studies were tested in a seated position, and this is a confounding factor that affects the hemodynamic responses after exercise (161,162).

After isokinetic resistance exercise, there was not enough activation of histamine H_1 and H_2 receptors in a design with a short protocol without leading to muscular failure that did not cause post-exercise hypotension (152). As was explained before, a minimum volume is necessary to provoke post-exercise hypotension. At the same time, it is probably necessary to elicit some muscular fatigue and a reduction in blood flow since these factors are contributors to the liberation or production of histamine or are contributors to the activation of histamine receptors (203–205). In this sense, it is known that the activation of these receptors provides protection against fatigue during exercise (152). Thus, protocols that differ in the appearance or not of muscular failure are needed to analyze the role that the histamine H_1 and H_2 receptors may have. As after aerobic exercise, nitric oxide appears to have little effect on post-exercise hypotension (206).

1.3.5. Applicability of the set configuration on post-exercise hypotension

To study the possible effect of muscle fatigue and failure on the onset of post-exercise hypotension, it is necessary to compare protocols equated in all the loading parameters, including the intensity of load. In this sense, set configuration here emerges as a variable that should be taken into account in post-exercise hypotension. Long set configurations, near or leading to muscular failure, provoke larger muscular contractions, a worsened blood flow and higher fatigue than short set configurations far from the onset of muscle failure. All these factors are contributors to the release or production of histamine or are contributors to the activation of the histamine receptors (203–205). In this sense, activation of histamine receptors provide protection against fatigue during exercise (152) and play a crucial role in the modulation of the metabolism during recovery (207).

Thus, it is plausible that long set configurations, especially set designs to failure, may help induce the onset of post-exercise hypotension due to these characteristics previously discussed. To test the hypothesis that reaching to muscular failure is a co-factor in the onset of post-exercise hypotension, it is necessary to compare protocols differing in the set design, this is, with or without muscular failure, while the rest of the parameters (i.e., intensity of load, volume, work-to-rest ratio) of the load remain equated. Also, to test the hypothesis that specifically fatigue, more than the fact of reaching muscle failure, may be responsible for the onset of post-exercise hypotension, it is necessary to compare protocols differing in the set configuration but without reaching to muscular failure. Additionally, the interaction with the type of exercise will be analyzed in protocols with and without muscular failure to analyze the possible implication of the lower limbs, since it is probably an important modulator of post-exercise hypotension (6,146,176).

It is hypothesized that set configuration leading to failure may be an important co-factor for the onset of post-exercise hypotension, maybe due to a substantial implication of the factors that are responsible for the release or production of histamine and/or due to the activation of the histamine receptors. Additionally, in protocols leading to failure and involving the lower limbs, post-exercise hypotension would be to a greater extent than protocols involving upper limbs. Otherwise, protocols without reaching muscular failure, even having longer set configurations, would not be important contributors because of this issue.

1.4. Effect of resistance exercise on perceived exertion

1.4.1. Perceived exertion

Perceived exertion scales are tools that serve to measure subjectively the intensity of effort, strain, discomfort, and/or fatigue that an individual feels while exercising (211). These scales are used, from a practical point of view and focused on resistance exercise, to know about the perceptual responses that are happening with certain loading parameters (35–37,212–214) and that are physiologically mediated (32).

The perceived exertion results from the complex integration of three different inputs to the central nervous system: Firstly, inputs centrally generated by forwarding neural signals, termed corollary discharges (208,209). Secondly, inputs peripherally produced by afferent feedback from the active organs, like the skeletal muscles implicated (210,211). And lastly, inputs of the information processing, like knowledge of the exercise task endpoint (212). The integrative response of all the inputs is summarized in an integer dependent on the scale used. In resistance exercise, the OMNI-RES scale is usually used which categorizes the perception of effort between a numerical response range between 0 and 10, and that is helped by pictorial and verbal descriptors that assist in anchoring the number (213). In this regard, its applicability has been focused on assessing the perceived exertion at the end of each exercise set (214,215). Nevertheless, there are not transcendent differences between scales since all are essentially the same (32,216). To a correct collection and use of the perceived exertion, some steps must be followed (217), such as explaining properly the instructions (213), establishing anchoring (35), and selecting the suitability of the overall perception or the active muscles perception (218). If this is properly performed, perceived exertion is actually able to distinguish numerous loading parameters (35–37,212–214) and is related somehow with certain physiological and neuromuscular parameters (213,219,220), as will be explained below.

1.4.2. Control of perceived exertion during resistance exercise

During resistance exercise, there is a progressive increase of perceived exertion with new repetitions (213,221) and sets (36) that leads to a maximal perceived exertion close or leading to muscular failure (222,223). Also, perceived exertion is determined by the intensity of load used (35,218,224) and modulated by the rest allowed between sets and exercises (37).

The control of the values obtained and their evolution would allow the possibility to regulate the resistance exercise session. These changes of the loading parameters and thus the possible physiological processes that are related may have different applications during a single workout of resistance exercise session and across the process of strength training. For example, regarding an individual work out of resistance exercise, a fixed perceived exertion value (e.g., 4) may correspond to a certain percentage of 1RM (225) or a range of the repetition maximum continuum (226). Besides, the perceived exertion may reasonably estimate the 1RM through progressive loads and their corresponding perceived exertion value (227) and is an indicator of the repetitions that can be performed prior to muscular failure (222).

Regarding the strength training process, perceived exertion may control the suitability of the intensity of load used since the values of perceived exertion are progressively descending when the 1RM is increasing (228), and the same perceived exertion value is obtained with a new absolute load after a period of training (229). Also, the perceived exertion may detect, with the help of other parameters of the session, negative processes that lead to illness and overtraining (33). Al last, it is possible that lower mean values of perceived exertion may lead to a higher middle-term adherence to exercise since a negative correlation was observed between both parameters (34).

1.4.3. Effect of resistance exercise on perceived exertion

As was explained above, in resistance exercise there is a progressive increase of perceived exertion with new repetitions (213,221) and with new sets (36), leading to the maximal perceived exertion value close or leading to muscular failure (222,223). In addition, several loading parameters, which are often related to each other somehow, determine the perceived exertion response:

The intensity of load used determines the perceived exertion. Each individual repetition (35,218) or group of repetitions (224) of an intensity of load have their own perceived exertion value, with higher values of perceived exertion with each higher intensity of load used (35,218,224). Nevertheless, this is not a universal finding (226).

Besides, there are increments of perceived exertion with each new set as observed after the first repetitions (36,37,230) or at the end of the new set (231). This is true in disregard of the intensity of load used, as long as the repetitions performed with each set (36,231) or the rest allowed between sets are fatiguing enough (37). In addition, short sets with an inter-repetition rest design also leads to an increase in perceived exertion (232). This increment in perceived exertion with sets was also observed within each new exercise performed in a resistance training session, showing a partial recovery in perceived exertion with each new exercise and a progressive increase with consecutive sets of the new exercise performed (219).

Additionally, the rest interval (37,233) and the type of exercise (234,235) performed might be modulators of the perceived exertion. On the rest interval, it is known that the rest between sets determines the perceived exertion in the first repetition of the next set (37), and probably at the end of the set (233), with higher values when the rests are shorter (37,233). Thus, this effect of the rest interval was also observed in short sets with an inter-repetition rest design, with lower values when the rest between repetitions is large enough (232).

On the type of exercise prescribed, their features can also affect the perceived exertion (234,235). Previously, discrepancies have been observed regarding the type of exercise performed, suggesting that the muscle mass implicated in the exercise could cause increases (234), no differences (36) or decreases (235) in the perceived exertion between exercises. The effect of the type of exercise and their characteristics, as the muscle mass implicated, should be elucidated for a better control of the effect of resistance exercise on perceived exertion.

The occurrence of muscular failure is a great determinant of the perceived exertion (236,237). Completing the maximum numbers of repetitions that can be performed in a set (i.e., RM or maximal. An intensity of effort of 100%) should be interpreted as a nearly maximum or maximum perceived exertion (i.e., 9-10 values) by the participant, due to anchoring (238). In this regard, perceived exertion is not usually capable of distinguishing between protocols differing in intensity of load (49,237,239), or rest (215,230,237) but that are leading their sets to muscular failure. Nevertheless, is is probably able to distinguish successive sets when these are leading to muscular failure (240,241).

In protocols to muscular failure, the total volume lifted seems to be the major determinant of perceived exertion (237,242–244). In this sense, designs with an equated volume lifted after an exercise (237,243) or a group of exercises (244) have shown comparable values of perceived exertion, despite the fact that were not equated in intensity of load (237,243) or rest (243). In addition, a previous study has shown that in protocols to failure, a design with a low intensity load but with a large volume performed, had higher values of perceived exertion in comparison with a design with a high intensity of load (242). All together, this suggests that in these kinds of protocols total volume lifted is more important that the intensity of load used (237,242,243).

However, in protocols without leading to muscular failure, the intensity of load used appears to be a major determinant of the perceived exertion (220,237,245,246), since individual sets (220,245,246) or consecutive sets (237) in protocols differing in intensity of load but with all of the rest of the parameters equated showed higher values of perceived exertion in the protocol with higher intensity of load used. When some determinants of perceived exertion in protocols leading (244) and without leading to muscular failure (237) are known, like some modulators (232,242), the extent of how these variables influence the perceived exertion are still unknown.

Previous studies have shown that perceived exertion during resistance exercise may be related to several physiological and neuromuscular variables, such as lactate (213,219) and cortisol production (219), or muscle activity (220). That relation suggests that some physiological and neuromuscular variables may determine in part the perceived exertion (213,219,220). In this sense, physiological and neuromuscular responses must be a consequence of the loading parameters used, which explains the perceptive responses observed (32).

Lactate production was observed to be positively correlated with perceived exertion (213,219), despite that in other studies this correlation was not observed (245,247). When differences in methodology may account in part for these discrepancies, the changes in lactate production are usually mirrored by the perceived exertion, even in total (213,248–251) or at least in part (245,252).

Cortisol is a neuroendocrine marker that augments due to physical or mental stress. Despite that only one article reported a positive association between perceived exertion and salivary cortisol (219), the perceived exertion during resistance exercise mirrors the production of cortisol after the session (219,253,254).

Finally, muscle activity refers to the active motor units and/or the firing frequency in which the motor units fire. The perceived exertion is functionally related somehow to the activity of the muscle, suggesting that the physiological processes related to their activation act as a mediator of the perceived exertion: When studies compared protocols differing in the intensity of load, a positive correlation between perceived exertion and muscle activity was observed (220). Moreover, perceived exertion and muscle activity behaved in a corresponding manner between protocols differing in load (245,246) and rest (247).

Taking all this into account, despite that some physiological and neuromuscular variables partially regulate the perceived exertion (213,219,220), as observed by alterations in the loading parameters (32), it is premature to hypothesize about how and in what extent these variables affect the perception of effort.

1.4.4. Applicability of the set configuration on perceived exertion

While some loading parameters such as the intensity of load (35,218,224), the volume (231), and the rest (37) determine and modulate the perceived exertion, the effects of other parameters are not established yet. In this regard, it is known that the duration of the repetitions (255) and the length of the set (213) also influence perceived exertion. Besides, both variables are logically related since the lower velocity observed at the end of longer sets means repetitions of more duration when the repetitions of the set are not paced externally (73).

The problem here emerges when other parameters want to be taken into account, such as the work-to-rest ratio. The work-to-rest ratio refers to the total work performed in relation with the total rest selected. As was previously explained, lower work-to-rest ratios (i.e., more rest in relation to the work done) as explained by differences in total rest between sets elicit lower values of perceived exertion (37,233). Also, the same finding was observed with inter-repetitions rest sets differing in rest between repetitions (232).

The study of the set configuration brings a unique contribution to this issue, since all the loading parameters are perfectly matched, exclusively allowing changes in the length of the set. One previous approach was made comparing protocols with the same intensity of load and also equated in the work-to-rest ratio (233). However, a percentage of the 1RM was chosen, not taking into account the maximum number of repetitions that can be performed and thus selecting a fixed number of repetitions. This may be a weakness in the design since the number of repetitions that can be done in a set is individual-dependent (45–50) and the remoteness and closeness to the muscular failure of each individual is a cofounding factor in relation to perceived exertion (237,242–244).

The design proposed may help to elucidate the effect of resistance exercise on perceived exertion when is prescribed through set configuration; which will be explained, to the best of our knowledge, for the first time. Also, their interaction with the type of exercise will be analyzed, as a trial to understand deeper the effects of set configuration on the perception of effort. As a consequence, this also might aid to understand the effect of the length of the set and the effect of the work-to-rest ratio in different exercises when all the loading parameters are equated. All this can contribute to the body of knowledge in perceived exertion, making the perceived exertion scales truly tools to prescribe resistance exercise based on the perception on the individual.

It is hypothesized that longer set configuration may cause higher values of perceived exertion in comparison with shorter set configurations. In addition, the interaction with the type of exercise performed would show higher values when the exercise is performed with more muscle mass implicated in comparison with exercises with less muscle mass involved. Finally, taking into account the difficulties of the scales of perceived exertion to discern between protocols leading to muscular failure, designs with the same configuration of the set but with different exercises would be not able to distinguish between set designs leading to failure, but would be able to discriminate between designs with other set configurations.

If this were true, the lower vales obtained in perceived exertion with shorter set configurations could have direct practical applications in training while maintaining the strength gains (70,83,92,93): it would allow to vary or reduce the loading parameters of the session that can lead to illness and overtraining (33) and facilitate a higher middle-term adherence to exercise (33), as was previously pointed out.

2. Purposes and hypotheses

Study I: A shorter set reduces the loss of cardiac autonomic and baroreflex control after resistance exercise.

Purpose

To analyze the effect of submaximal set configurations of resistance exercise on post-exercise autonomic and baroreflex control and in blood pressure.

Hypothesis

Longer set configurations without leading to failure would produce a higher cardiac vagal withdrawal and loss in BRS, and a large reduction in post-exercise blood pressure in comparison with shorter set configurations.

Study II: Exercise type affects cardiac vagal autonomic recovery after a resistance training session.

Purpose

To analyze the effect of maximal versus submaximal set configurations of resistance exercise on post-exercise cardiac vagal control and blood pressure and their interaction with the type of exercise.

Hypothesis

The maximal set configuration and the exercise with more muscle mass involved would produce a higher cardiac vagal withdrawal and a large reduction of blood pressure after exercise in comparison with a submaximal short set configurations and an exercise with less muscle mass involved.

Study III: Effects of set configuration of resistance exercise on perceived exertion.

Purpose

To analyze the effect set configurations of resistance exercise on perceived exertion and their interaction with the type of exercise.

Hypothesis

The long set configuration and the exercise with more muscle mass involved would produce a higher perceived exertion in comparison with a short set configuration and an exercise with less muscle mass involved.

3. Study I

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.Wetested 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.

3.1. Introduction

Long-term resistance training has been shown to be beneficial for prevention and improvement of musculoskeletal, metabolic or cardiovascular conditions (1). In addition, resistance training improves several markers of cardiac autonomic control in both healthy (256) and diseased individuals (257).

HRV and 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 (12,98). A resistance training session induced changes in HRV and HRC suggesting a transient reduction in cardiac vagal control after exercise (27). Also, a resistance exercise session may produce a decrement in BRS (5,16). Nevertheless, the effects of the loading parameters of resistance exercise on autonomic control and BRS are not fully understood (27).

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 (103) and volume (30), meanwhile others have not confirmed these findings (13,171). 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 (80) and modulates the metabolic involvement in the session (77).

Shorter set configurations, termed cluster training or inter-repetition rest training (64), result in a higher velocity and a lower glycolytic metabolism (77) 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 (92). 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 (103) and BRS (15) 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 (102). 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 load, 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 load, 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 this parameters (i.e., load, total volume, total rest and therefore the work-to-rest ratio) are strictly equated with except of 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.

3.2. Methods

17 healthy adults participated in this study, with at least six months of experience lifting weights two or three times/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 (Appendix B) and were informed they could withdraw at any time. The characteristics of the participants are shown in Table 1.

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 hours and fast for three hours 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.

Table 1. 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 heart rate (beats/min)	61± 14
Resting systolic blood pressure (mmHg)	116 ± 9
Resting diastolic blood pressure (mmHg)	68 ± 7
Resting mean arterial pressure (mmHg)	87 ± 7
10 RM in Leg Press (kg)	211 ± 45

Data displayed as means ± SD

Orientation sessions. Participants completed three familiarization sessions in which they were instructed on how to perform the leg press exercise programs 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 reach a 90º 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 (258). 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 protocol were going to perform until the beginning of the session. For each exercise session, the loading parameters (i.e., load, 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%]) and 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 minutes. Sessions were separated by at least 72 hours 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 5.

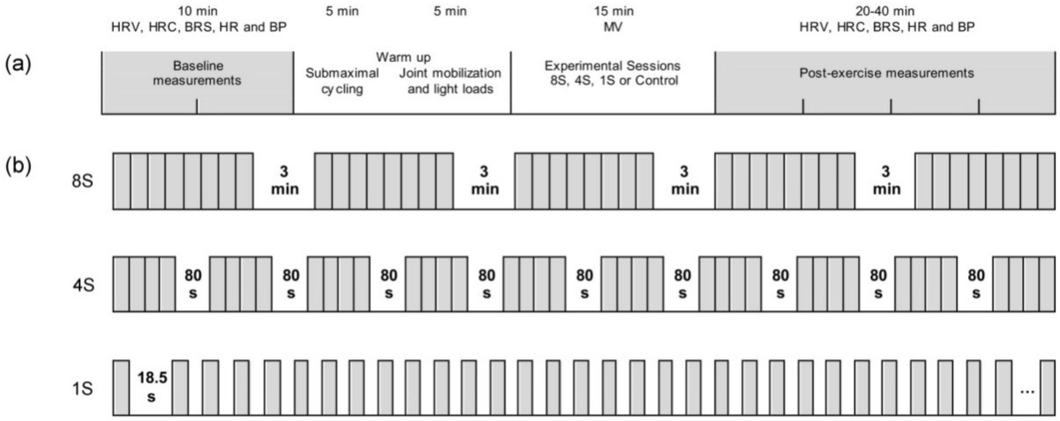

Figure 5. Schematic representation of the sessions. (a) Graphical simplification of the entire protocols design. HRV, heart rate variability; HRC, heart rate complexity; BRS, baroreflex sensitivity; HR, heart rate; BP, blood pressure; MV, mean velocity. (b) Representation of the experimental sessions. 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

Physiological recording. A Task Force Monitor (CNSystems, Austria) was used for continuous monitoring of the HR and BP. HR was obtained by a three lead electrocardiogram (ECG) 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 Task Force Monitor 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 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 (27).

Physiological assessment. We analyzed the heart rate (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 and frequency domain 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 (98). 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 parasympahtetic activities (98).

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 (259). Epochs of 5 min were used as recommended by guidelines for HRV analysis during short-term recording (98). Analysis of HRC was performed with Sample entropy (SampEn). Meanwhile 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 heart rate dynamics (99). 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 find specific patterns in a range from 0-2, being less predictive (i.e., complex) when values are close to 2 (260). 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 of the time series and epochs of 5 min were used to the analysis following the suggestions published elsewhere (12).

Automatic artifact 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). Artifact correction never exceeded the 3% of the signal.

BRS was calculated using the sequence method (124) with the Task Force Monitor 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 analyze BRS after resistance exercise (15,16).

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 (71). 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 ± standard deviation (SD). Intra-Class Correlation Coefficient (ICC) with Single Measure Intra-Class correlation was used to test the reliability of the 10RM (ICC= 0.989). A 1-way repeated measures analysis of variance (ANOVA) was used to evaluate the effect of session (1S, 4S or 8S) on the averaged mean velocity of every repetition. A 2-way repeated measures ANOVA (session x 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 were log transformed. Post-hoc comparisons were performed with Bonferroni correction. A $p \leq 0.05$ was established as statistical significance. The data were analyzed 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-β) 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.

3.3. Results

Autonomic and baroreflex data. Autonomic and reflex data are shown in Table 2. Values before exercise were similar between protocols for all variables (p>0.05). For Ln of RMSSD, main effects for session ($F_{3, 48}$= 5.491 p=0.003) and time ($F_{4, 64}$= 7.732 p=0.004) were observed. The main effect of session revealed that 8S was significantly lower than Control session (p=0.01). The main effect of time showed that the epoch of 20-25 min (p=0.028) was significantly lower than the Pre values. Also, an interaction between session and time was observed ($F_{12, 192}$= 13.580, p<0.001). 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>0.05).

For Ln of HF, main effects for session ($F_{3,48}$= 3.582 p=0.02) and time ($F_{4,64}$= 6.429 p=0.004) 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=0.028) was lower than the Pre values. Besides, an interaction between session and time was detected ($F_{12,192}$= 4.556, p=0.003). 8S had lower values of HF in comparison with the Control session and Pre values, for the period 20-35 min. 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>0.05).

SampEn showed a main effect of session ($F_{3,48}$= 5.115, p=0.012). Pairwise comparison revealed no differences among protocols. There was not interaction between session and time (p>0.05).

For Ln of BRS, main effects for session ($F_{3,45}$= 4.756 p=0.006) and time ($F_{2,30}$= 15.385 p<0.001) were observed. The main effect for session revealed a lower BRS values in 8S in comparison with Control session (p=0.007). The main effect of time showed lower values of BRS for all the postexercise period. The p-values of BRS for the periods were: 20-30 min (p=0.002) and 30-40 min (p=0.026) respect to baseline data. In addition to this, an interaction between session and time was observed ($F_{6,90}$= 5.902, p=0.002). 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=0.016). 4S had lower values of LF in comparison to the Control session in the period 20-25 min (p=0.038) and in comparison to the 1S in the epoch 25-30 min (p=0.045). No main effects were observed for this variable (p>0.05).

Table 2. 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†*	

†Different vs. C (p<0.05). ‡ Different vs. 1S (p<0.05) * Different vs. Pre (p<0.05)

For Ln of LF/HF, a main effect of time was observed ($F_{4, 64}$= 7.932, p<0.001). The postexercise epochs of 20-25 (p=0.006), 30-35 (p=0.006) and 35-40 min (p=0.026) were higher than the Pre values. Neither the main effect for session nor the interaction between session and time were significant (p>0.05).

LFnu showed a main effect of time ($F_{4, 64}$= 8.105, p<0.001). Pairwise comparison showed higher values in the postexercise periods of 20-25 (p=0.006), 30-35 (p=0.008) and 35-40 min (p=0.023) in comparison to Pre values. There was not a main effect of session or an interaction between session and time (p>0.05).

Hemodynamic data. Hemodynamic data are shown in Table 3. For SBP, DBP and MAP, no main effects or interactions were observed among protocols (p>0.05).

Table 3. Hemodynamic responses across sessions (n=17)

	Pre	20-25	25-30	30-35	35-40
Systolic blood pressure (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
Diastolic blood pressure (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
Mean arterial pressure (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

Data displayed as means ± SD

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=0.016) and between 4S and 8S (p=0.045), with lower values for 8S.

3.4. 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 (103,107). 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 correlates with lactate production, with a higher lactate production in protocols with a lower velocity (71). On the other hand, set configuration similar to our 1S is characterized by higher velocity and lower glycolytic involvement than a traditional configuration (77). 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 (4,5,11–13). However, significant differences between resistance exercise protocols are scarce. Some studies have shown that intensity (103) and volume (30) may affect the vagal control of the heart, meanwhile others have not confirmed these findings (13,171). Contrary to our current data, a recent study (102) 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 a ordinary active population in our study), since it has been reported that volume (30) and resistance-training experience (13) 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 (102). 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 (17). 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 (99). In this sense, previous studies have reported reductions in complexity without changes in the variability parameters after a resistance session (12,13) 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 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 (5,15). In our study, longer sets had a lower velocity than the shorter sets, and the loss of velocity indicates neuromuscular fatigue (71). 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 (12), 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 to BRS (5,15). 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 what suggests that set configuration is useful in order to regulate the loss in the reflex control of the heart after resistance exercise.

These impact 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 (17). Also, prognosis studies revealed that reductions in both cardiac vagal modulation and BRS are associated with myocardial ischemia and sudden cardiac death (97).

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 (6,30). Our results agree with previous studies in which similar protocols were insufficient to provoke changes in blood pressure after resistance exercise (6).

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 gender 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.

3.5. Conclusions

Our study suggests that 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.

4. Study II

Exercise type affects cardiac vagal autonomic recovery after a resistance training session

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Abstract

Resistance training session involving different exercises and set configurations may affect the acute cardiovascular recovery pattern. We explored the interaction between exercise type and set configuration on the postexercise cardiovagal withdrawal measured by heart rate variability and their hypotensive effect. Thirteen healthy participants (10RM parallel squat: 91±13, bench press: 56±10 kg) performed six sessions corresponding to two exercises (Parallel squat vs Bench press), two set configurations (Failure session vs Inter-repetition rest session) and a Control session of each exercise. Load (10RM), volume (5 sets) and rest (720 sec) were equated between exercises and set configurations. Parallel squat produced higher reductions in cardiovagal recovery versus Bench press ($p=0.001$). These differences were dependent on the set configuration, with lower values in Parallel squat versus Bench press for Inter-repetition rest session (1.816±0.711 vs 2.399±0.739 Ln HF/IRR^2 $x10^4$, $p=0.002$), but not for Failure session (1.647±0.904 vs 1.808±0.703 Ln HF/IRR^2 $x10^4$, $p>0.05$). Set configuration affected the cardiovagal recovery, with lower values in Failure session in comparison with Inter-repetition rest ($p=0.027$) and Control session ($p=0.022$). Postexercise hypotension was not dependent on the exercise type ($p>0.05$) but was dependent on the set configuration, with lower values of systolic ($p=0.004$) and diastolic ($p=0.011$) blood pressure after the Failure session but not after an Inter-repetition rest session in comparison with the Control session ($p>0.05$). These results suggest that the exercise type and an Inter-repetition rest design could blunt the decrease of vagal activity after exercise while exercising to muscular failure may contribute to the onset of postexercise hypotension.

4.1. Introduction

Resistance training is considered a promising intervention to prevent and improve several musculoskeletal, metabolic and cardiovascular conditions (1). Nevertheless, previous studies have revealed through HRV that resistance training seems not to improve the autonomic control of the heart (27). Additionally, resistance exercise session provokes an acute reduction in the cardiac vagal control (4,5,11–13) that can be interpreted as a transient harmful effect in diseased individuals since 30 min after exercise exists an increased probability of suffering a sudden cardiac death due to a decreased vagal activity (17). In this sense, the effect of the characteristics of the session and the loading parameters of resistance exercise are not adequately understood (27). Coaches and practitioners should know the precise effects of the training factors that affect the cardiac control after resistance exercise to modulate the cardiac impact and prescribe exercise in an accurate way. Especially, resistance exercise should be prescribed in a riskless manner in certain individuals with cardiovascular risk. In order to solve this issue, these effects on the cardiac control should be fully elucidated. In this regard, cardiac vagal control after a resistance training session has been shown to be affected by intensity (103,105) and volume (30), although others have not confirmed these findings (13,171).

Other factors that may affect the cardiac control after resistance exercise are, on the one hand, the type and features of the exercise used, and on the other hand, the set configuration employed. The type and features of the exercise used refer to the muscle mass of the exercises performed and the body postures associate in those exercises. In this sense, exercises with a higher muscle mass involvement produce higher lactatemia (71), and the glycolytic involvement is related to the cardiac vagal withdrawal (103,107). To our knowledge, only one study analyzed the effect of the exercises type (i.e. upper versus lower limbs) on the vagal control without detect significant differences between protocols (13). Nevertheless, the exercises were performed to muscular failure and the position of the exercises was always seated, variables that may have eliminated the differences between protocols.

Besides the effect of exercise type, the set configuration could also affect the vagal recovery to resistance exercise. Set configuration refers to the repetitions actually performed with regard to the maximum possible number of repetitions in a set. Set configuration is closely associated with intensity and volume, and affects the mechanical performance and the metabolic response to exercise (71,77). Short set configurations as the inter-repetition rest design (65,77) result in a higher mechanical performance and lower involvement of the glycolytic energy system than long set configurations, close or leading to muscular failure (71). As was explained before, the involvement of the glycolytic energy system during resistance exercise seems to be related to the changes in the postexercise cardiac vagal control (103,107). However, only one study have explored the effects of the set configuration on the cardiac vagal control (102). That study showed that the reduction of the vagal control of the heart after a resistance exercise was similar between sets performed to muscular failure and an inter-repetition rest set configuration. However, in that study only parallel squat exercise was analyzed and thus, it remains to be explore whether those results can be replicated with different exercises. It is likely that these inconsistent results could result from the interaction between the set configurations and the exercise used in that study.

In contrast with the possible harmful effect that means the transient reduction in the vagal control of the heart, postexercise hypotension is a positive acute effect of resistance exercise that has been extensively reported in the literature (20). A recent study has shown a relationship between the postexercise hypotension caused by resistance exercise and long-term blood pressure reduction at rest (141), making exercise as an interesting non-pharmacological therapy to reduce blood pressure. Nevertheless, the loading parameters that affect postexercise hypotension are not completely established. In his sense, it is known that the volume performed is a key factor to induce postexercise hypotension (6,30). However, the impact of the exercise type and the muscular failure on the postexercise hypotension is not conclusive: About the exercise type, some studies suggest that hypotensive effect depends on muscle mass engaged in the exercise (6,176), while others did not support this finding (187,261). About muscular failure, postexercise hypotension was observed in this kind of protocol in comparison with a non-failure design (11), suggesting that muscular failure may be an important contributor to this effect. However, the load between protocols were not equated, something that may affect the results (106).

Therefore, the main objective of this study was to explore the impact of the exercise type and set configuration on the recovery pattern of cardiac vagal modulation and postexercise hypotension. In this regard, our aim is to identify the exercise type and set configuration in which the cardiac autonomic control is less affected, and the possible interaction between them. This may have practical applications to prescribing resistance exercise in individuals at cardiovascular risk. Also, our aim is to understand the implication of the exercise type and the set configuration with muscular failure when all the loading parameters are equated, something that may help to understand the onset or increase of the postexercise hypotension in order to prescribe resistance exercise as a non-pharmacological therapy to reduce blood pressure in hypertensive individuals.

In order to do that, participants performed six experimental sessions, corresponding to the combination of two types of exercises (bench press and parallel squat) with two set configurations (sets leading to muscular failure and an inter-repetition rest design) and two control sessions, one for each exercise. All sessions had the same load, volume and work-to-rest ratio, allowing the comparison between exercises and set configurations. Our hypothesis is that parallel squat and long set configurations leading to muscular failure would produce higher levels of cardiac vagal withdrawal and postexercise hypotension than a short set configurations as an inter-repetition rest design.

4.2. Methods

Experimental approach to the problem. A repeated measures design was performed in order to test the impact of both exercise type and set configuration on the acute changes of cardiovagal control and blood pressure after a resistance training session. Thus, participants performed six experimental sessions corresponding to two exercise types (parallel squat and bench press) and three experimental protocols (control session, session to muscular failure and a an inter-repetition rest session). All exercising sessions had the same load, volume and work-to-rest ratio in order to properly identify main effect and interaction between exercise type and set configurations.

*Participant*s**.** Thirteen normotensive males sport science students, with at least 6 months of experience in resistance training lifting completed this study. They were screened and excluded if they had prior history of cardiovascular disease, orthopedic pathology, or illness. All participants signed an informed consent form (Appendix B) and were informed they could withdraw from the study at any time. The study was approved by the local Institutional Ethics Committee. The physical, cardiovascular and functional characteristics of the participants are shown in Table 4.

Table 4. Physical, cardiovascular and functional characteristics of the participants (n=13)

Age (yr)	23 ±3
Height (m)	1.76±0.05
Body mass (kg)	72.1±5.8
Body mass index (kg/m^2)	23.4±1.2
Systolic blood pressure (mmHg)	113±6
Diastolic blood pressure (mmHg)	63±6
Mean arterial pressure (mmHg)	79±6
Heart rate (bpm)	54±6
10RM Press (kg)	56±10
10RM Squat (kg)	91±13

Data displayed as means ± SD

Procedures. A repeated-measures design was used in this study. Participants attended 11 days to the laboratory: 5 for orientation procedures and 6 for experimental protocols. During the course of the experiment testing, participants were asked to refrain from alcohol, caffeine, nutritional supplements, nicotine, and exercise for 24 hours and fast for the three hours prior to the beginning of the sessions. The warm-up of each session was composed by 5 minutes of submaximal treadmill exercise, 5 minutes of joint mobilization and calisthenics, and 2 sets of 10 repetitions with light loads.

Orientation sessions. Participants were instructed on how to properly perform bench press and parallel squat in three familiarization sessions that consisted in of five progressive submaximal sets with 10 repetitions. In the following two sessions, 10 RM was tested to establish reliability. Both exercises were performed in a Smith Machine (Life Fitness, Brunswick Corporation, USA). The bench press exercise was performed with the participants starting with the elbows extended. Then, the bar was lowered to the chest in a controlled manner. Approximately 1 sec. was waited before the start of the concentric phase to eliminate the rebound effect and to obtain more consistent measures. Participants performed the concentric phase as fast as possible. The grip width was set at 130% of biacromial breadth. The parallel squat exercise was performed starting from the upright position with the knees extended, the feet parallel and placed shoulder width apart, and the barbell resting across the back. Participants then lowered in a controlled manner until the thigh was horizontal to the floor with the knees at approximately 90º of flexion. Finally, participants recovered the initial position, performing each repetition as fast as possible. The same researcher provided verbal encouragement in order to incite maximal effort by the participants.

A previously reported protocol was employed to obtain the 10RM load (258). 10RM was defined as the load that a participant was able to lift properly 10 times, but not 11. Participants performed no more than 5 attempts on each exercise with a rest interval of 2–5 min. between attempts.

Experimental sessions. Participants completed 4 sessions corresponding to the combination of two types of exercises (Bench press and Parallel squat) and two set configurations: Failure session and Inter-repetition rest session. Additionally, two Control sessions were conducted as a reference for Bench press and Parallel squat protocols.

Failure session consisted of 5 sets to failure with the 10RM load and with 180 sec. of rest between sets (i.e. 720 sec. of total resting time). Inter-repetition rest session consisted of the same number of repetitions completed in the Failure session, but with the total resting time (i.e. 720 sec.) distributed between each repetition. Thus, the work-to-rest ratio was equated between set configurations while the load (i.e., 10RM), volume (maximum number of repetitions performed in 5 sets) and rest (720 sec.) were similar between exercises. The Control session consisted in maintaining the body position of the exercises (i.e. lying on a bench for bench press and standing for parallel squat) during 15 min, but without performing any exercise. The order of exercises and control sessions were randomized. However, since the number of repetitions in the Inter-repetition rest session depended on the volume completed in the Failure session, it was not possible to randomize the order of the set configurations.

The number of repetitions performed during the Failure session were different across participant and thus, the rest intervals between each repetition were individualized during the Inter-repetition rest session (262). Participants completed all repetitions in the Inter-repetition rest sessions without muscular failure. The sessions were separated by at least 72 hr. and were performed at approximately the same hour of the day (±1 hour) by each participant.

Physiological recording. A portable cardiac monitor (Polar RS800CX, Kempele, Finland) was used for beat-by-beat heart rate (HR) recording. An oscillometric device (Omron MIT Elite Plus, Kyoto, Japan) with proper sized cuff was employed for registering SBP and DBP before and after every session. Data were obtained 10 min prior and in the period 20-40 min after the exercise with the participant seated and breathing spontaneously. Data were recorded in a seated position at the end of a 20 min resting period after exercise to reduce the effect of the increased respiratory rate on the variables (263).

Data analyses. HRV was used to estimate the vagal autonomic modulation of the heart. Fast Fourier Transformation method was selected in order to analyze the high frequency activity (HF, 0.15-0.4 Hz) in absolute units. HF is used as an indicator of cardiac vagal modulation (264). Kubios HRV software v2.1 (The Biomedical Signal and Medical Imaging Analysis Group, University of Kuopio, Kuopio, Finland) was used to analyze beat to beat intervals series with an automatic artifact correction (i.e., medium correction level). To weaken the HRV dependence on HR, HF was divided by the squared R-R interval (IRR^2) of each epoch (265,266). Thereafter, the resultant of this division was log transformed since HF did not achieve normality, and multiplied by 10.000 to achieve positive parameters and facilitate the understanding of the results. This change in the scale does not modify the mathematical properties of the values. Prior to the warm-up, a 10 minutes period were recorded with the participants resting in a seated position. HRV was obtained in the last 5 minutes, while SBP and DBP were assessed at minute 8 and 10 of this period. After exercise, variables were obtained in epochs of 5 min across the 20-40 minutes for HRV and at minutes 20, 25, 30, 35 and 40 minutes postexercise for SBP and DBP.

Measurement of dynamic performance. A dynamic measurement device was used (T-Force System, Ergotech, Spain) in order to evaluate the mechanical performance as an indicator of neuromuscular fatigue (267). The propulsive part of the concentric phase of each repetition was analyzed and the mean propulsive velocity (MPV) was averaged to the entire session. The propulsive part is the portion during which acceleration is greater than the acceleration due to gravity (267). To ensure that there was no learning effect due to the non-randomization of the sets, the fastest repetitions of every set configuration and each exercise were compared, since the velocity of each load determines the relative load of the exercise (42).

Statistical analyses. Descriptive statistics were calculated as mean ± standard deviation (SD). To establish the reliability of the 10RM test, the Intra-class Correlation Coefficients (ICC) with Single Measure Intra-Class correlation was determined (ICC = 0.98 and 0.97 for bench press and parallel squat, respectively). Shapiro–Wilk test was used to test normal distribution of parameters. Data were log transformed (Ln) in the case that normality assumption was violated. A paired t-test was used to compare within exercises the fastest repetitions of every set configuration (Failure session vs. Inter-repetition rest session). A 2-way repeated-measures ANOVA (exercise × set) was performed to compare the number of repetitions performed across the 5 sets within Failure sessions. A 3-way repeated measures ANOVA (exercise x protocol x time) was performed to evaluate the effect and interaction between Exercise (Bench press or Parallel squat), Protocol (Failure session, Inter-repetition rest session and Control session) and Time (Pre and 20-25, 25-30, 30-35, 35-40 min epochs for HRV; and 20, 25, 30, 35, and 40 min moments for SBP and DBP). Multiple comparisons with Bonferroni correction were performed when necessary. Analysis of the Effect Size was performed with the partial Eta squared (η_p^2). Statistical significance was established with a p value of ≤0.05. The data were analyzed 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-β) of a repeated measures ANOVA with 4, 5 and 6 measurements for a sample size of 13, a correlation among repeated measures of 0.5 and a medium effect size (f=0.25) is 0.51, 0.56 and 0.62, respectively.

4.3. Results

Numbers of repetitions in Failure session were 32±5 and 34±6 repetitions for Bench press and Parallel squat, respectively. A significant effect of set was observed with a progressive decrease in the number of repetitions with each subsequent set ($F_{4,52}$= 63.256, $p< 0.001$). Neither main effect of exercise, nor significant interaction between exercises and sets was observed ($p>0.05$). Rest intervals between each repetition during Inter-repetition rest session were 23.6±4.1 and 22.9±4.6 sec. for Bench press and Parallel squat, respectively. To analyze the possible learning effect due to the non-randomization of the set configuration, the fastest repetition of the two set configurations of each exercise was compared. There were no differences in the velocity of the fastest repetition between set configurations nor for Bench press neither for Parallel squat ($p>0.05$).

Autonomic data. For Ln of HF/IRR^2 x 10^4, main effects were observed for Exercise ($F_{1,12}$= 9.803, p=0.009; η_p^2= 0.45), Protocol ($F_{2,24}$= 8.426, p=0.002; η_p^2= 0.413), and Time ($F_{4,48}$= 8.669; p=0.001. η_p^2= 0.419). Significant interactions were observed between Exercise and Time ($F_{4,48}$= 6.800, p=0.001; η_p^2= 0.362) and Protocol and Time ($F_{4,48}$= 9.625, p<0.001; η_p^2= 0.445) and Exercise and Protocol ($F_{2,24}$= 4.448, p=0.0023; η_p^2= 0.270).

The interaction between Exercise and Time (Figure 6) revealed differences between exercises for the period 20-35 min ($p\leq0.001$-0.012) with lower values for Parallel squat. Also, differences between moments were dependent on exercise. For Parallel squat, values were lower in the period 20-35 (p=0.003-0.044) respect to the pre-values; meanwhile for Bench press, differences were not observed between measurements. Additionally, no differences were observed in the pre values between exercises.

The interaction between Protocol and Time (Figure 7) of Ln of HF/IRR2 x 10^4 revealed differences between protocols for the post-exercise period. Control session was higher than Failure session for the entire post-exercise period: 20-40 ($p \leq 0.001$-0.017). Furthermore, differences versus the Pre values depended on the protocol. In Failure session, lower values were observed in the period 20-30 ($p=0.003$-0.004) in comparison with Pre values, while no differences were observed between moments in Control session or Inter-repetition rest session. Finally, no differences were observed in the pre values between exercises.

The interaction between Exercise and Protocol (Figure 8) of Ln of HF/IRR^2 x 10^4 revealed that differences were observed between exercises for Inter-repetition rest session, with lower values in Parallel squat in comparison with Bench press ($p=0.002$). Also, differences between protocols were observed and dependent on exercise. For Parallel squat, lower values were observed for Failure session ($p=0.008$) and Inter-repetition rest session ($p=0.037$) in comparison with Control session. However, for Bench press lower values were observed in Failure session respect to both Inter-repetition rest session ($p=0.027$) and Control session ($p=0.022$). Besides, no differences were observed between Control session and Inter-repetition rest session for bench press.

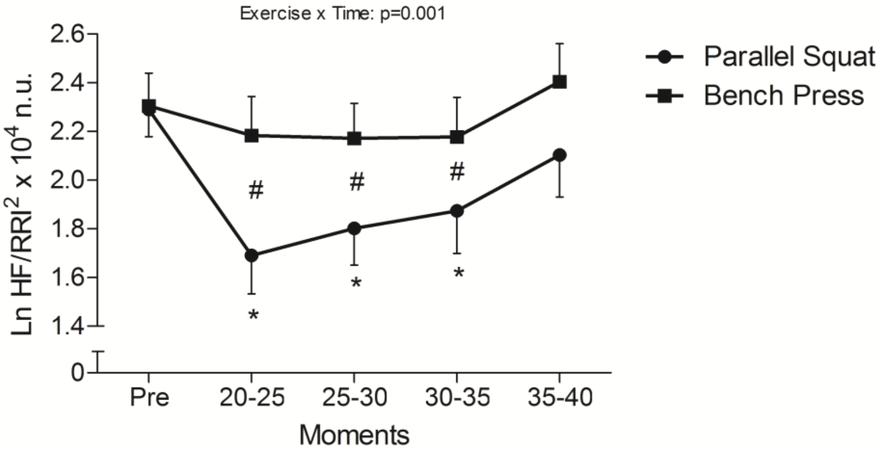


Figure 6. Interaction between Exercise and Time for the Ln of HF/IRR^2 x 10^4 (n=13)
In circles, Parallel Squat. In squares, Bench press
* Differences versus pre-values of the same exercise
\# Differences between different exercises
Data displayed as means ± SE

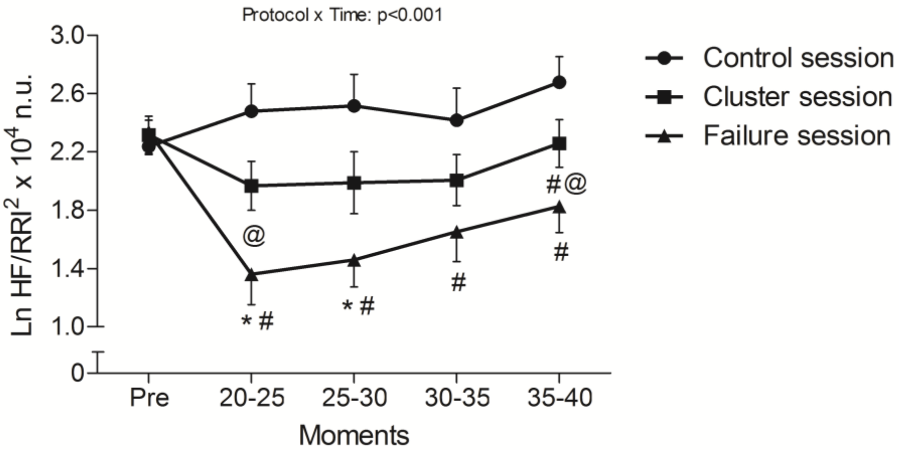


Figure 7. Interaction between Protocol and Time for the Ln of HF/IRR^2 x 10^4 (n=13)
In circles, Control session. In squares, Inter-repetition rest session
In triangles, Failure session
* Differences versus the pre-values of the same protocol
# Differences versus the Control session
@ Differences between Cluster session
Data displayed as means ± SE

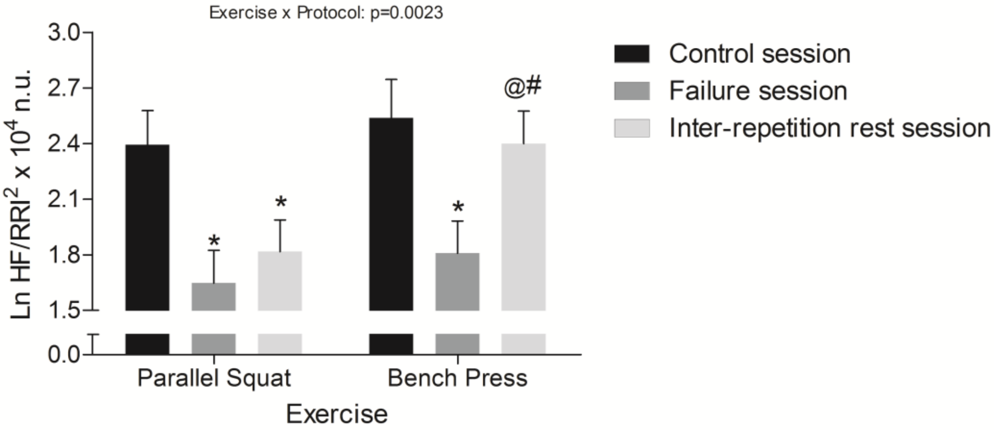
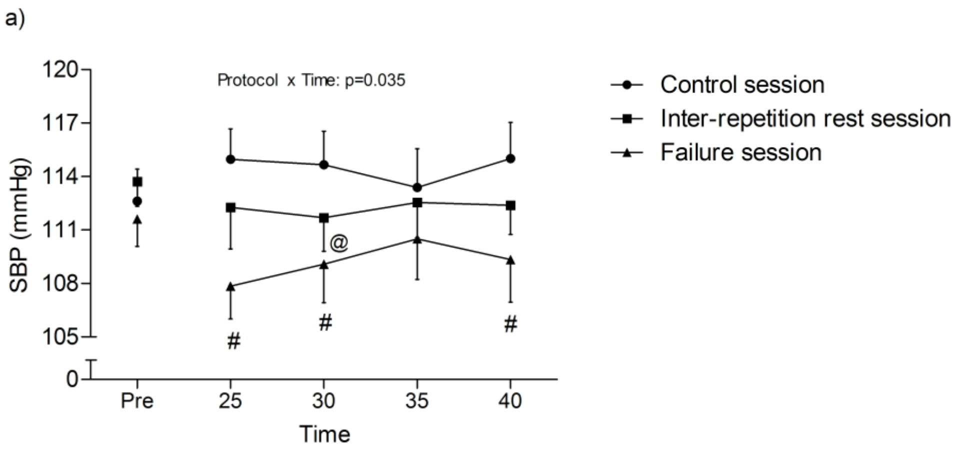


Figure 8. Interaction between Exercise and Protocol for the Ln of HF/IRR^2 x 10^4 (n=13)
In black, Control session. In dark gray, Failure session
In light gray, Inter-repetition rest session
* Differences versus the Control session
# Differences versus the Failure session of the Bench Press
@ Differences versus the Inter-repetition rest session of the Parallel Squat
Data displayed as means ± SE

Hemodynamic data. For SBP, a main effect for Protocol ($F_{2,\ 24}$= 10.429, p=0.001; η_p^2 = 0.465) was observed. Failure session was significant lower than Control session (p=0.004) and Inter-repetition rest session (p=0.008). Additionally there was a significant interaction between Protocol and Time ($F_{8,\ 96}$= 2.186, p=0.035; η_p^2= 0.154) (Figure 9a). Failure session was significantly lower than the Control session at 25 (p=0.006), 30 (p=0.009) and 40 min (p=0.017) after exercise. Failure session was also lower with respect to Inter-repetition rest session at 30 min (p=0.036). Pre-post comparisons were not significant for any session. Finally, differences between sessions at the baseline were not significant.

For DBP, a main effect for Protocol ($F_{2,\ 24}$= 7.232, p=0.003; η_p^2 = 0.376) was observed. Failure session was significant lower than control session (p=0.011). Additionally, there was a significant interaction between Protocol and Time ($F_{8,\ 96}$= 4.253, p=0.006; η_p^2 = 0.262) (Figure 9b). Failure session was significant lower than the Control session for the entire post-exercise period: 25 (p=0.034), 30 (p=0.02), 35 (p=0.001) and 40 min (p=0.009). Also, the Failure session was lower with respect to Inter-repetition rest session at 35 min after exercise cessation (p=0.05). In the Control session there was a significant increase in DBP for 30 (p=.002), 35 (p=0.011) and 40 min (p=0.014) moments respect to the baseline values. There were not differences between pre-post comparisons for any protocol. Differences in pre values were neither observed.

Dynamic performance measurements. MPV values showed a significant lower velocity in the Failure session in comparison with Inter-repetition rest session for both Parallel squat (0.33±0.4 $m.s^{-1}$ versus 0.41±0.1 $m.s^{-1}$, p=0.001) and Bench press (0.28±0.05 $m.s^{-1}$ versus 0.40±0.07 $m.s^{-1}$, p<0.001) exercises.



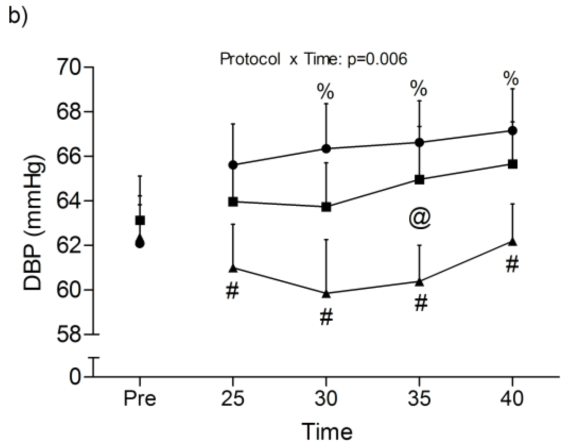


Figure 9. a) Interaction between Protocol and Time for SBP (a) and DBP (b) (n=13)
In circles, Control session
In squares, Inter-repetition rest session
In triangles, Failure session
Differences versus the Control session
@ Differences between Inter-repetition rest session and Failure session
% Significant higher versus pre-values
Data displayed as means ± SE

4.4. Discussion

In the current study we explore the effect of the exercise type, set configuration and their interaction on the postexercise cardiovagal withdrawal and the hypotensive effect. The main findings of this study were a) the type of exercise affected the cardiac vagal autonomic control after a resistance exercise, with less control after parallel squat in comparison with bench press exercise. b) Set configuration also affected the autonomic cardiac vagal control after exercise, with less control in Failure session in comparison with Inter-repetition rest session and Control session. c) Interactions between the type of exercise and the set configurations revealed that the cardiac vagal control is affected by both factors simultaneously. d) Systolic and diastolic blood pressure after failure sessions were decreased respect to control sessions.

Our study shows that when load, volume and work-to-rest ratio are equated, the type of exercise affects the pattern of recovery of the vagal autonomic control of the heart (Fig. 1). The lower reduction in the cardiac vagal control after Bench press in comparison with Parallel squat could be caused by the body positions and muscle masses involved in those exercises. The lying position during the bench press could facilitate the venous return and the cardiac filling of the ventricle during the preload, in comparison with the stand up position during the Parallel squat. In addition, the lower amount of muscle mass involved during a bench press exercise in comparison with a parallel squat could induce lower glycolytic involvement for the former.

Although lactate was not analyzed in the current study, a previous study reported higher levels of blood lactate concentration during squat in comparison with bench press with the same load (71). Therefore, we can speculate that the low glycolytic involvement during the bench press could blunt the loss of cardiac vagal control, since previous studies have suggested a relationship between cardiac vagal withdrawal and lactate levels both at rest with intravenous injection of lactate (108,268) and after resistance exercises (103,107). To the best of our knowledge, only one study analyzed the effect of the exercises type (i.e. upper versus lower limbs) on the cardiac vagal control without detect significant differences between protocols (13). However, both exercises were performed to muscular failure, causing a high fatigue that could overcome a plausible difference effect of each exercise. In the present study, when both exercises were performed to muscular failure, no differences were observed, which is in agreement with that study (13). These findings suggest that training to muscular failure may provoke a significant and comparable reduction in vagal control regardless of the type of exercise performed.

Set configuration also affects the pattern of recovery of the vagal autonomic control of the heart (Fig. 2). Our study shows that Failure session had a higher loss of vagal control in comparison with Control session and Inter-repetition rest session. Meanwhile, for Inter-repetition rest session the cardiac vagal control was scarcely affected. Nevertheless, when the type of exercise was taking into account, set configuration modulated cardiac vagal response for Bench press but not for Parallel squat (Fig. 3). In Bench press, Failure session induced less vagal control than Inter-rest repetition session. In Parallel squat, these differences were not observed. These results for parallel squat are coincident with a previous study comparing a session to muscular failure versus an inter-repetition rest session for the same exercise (102).

The differences observed for set configuration between bench press and parallel squat may be due to the type and features of the resistance exercise performed. It is possible that when protocols are performed in exercises that involve large muscle mass, the loss in cardiac vagal control is comparable regardless the set configuration used.

After resistance exercise there is a reduction in the vagal control of the heart (4,5,11–13), that may mean a transient harmful effect in individuals at cardiovascular risk since 30 min after exercise exists an augmented probability of suffering a sudden cardiac death due to a reduction in vagal activity (17). Therefore, the usefulness of controlling the loading parameters in order to minimize this reduction may be dependent on the exercise type, the set configuration and the interaction between them. It seems that exercises performed in a lying position, with less muscle mass implicated and done with a short set configuration as the inter-repetition rest design may reduce the loss of cardiac vagal control of the heart, guaranteeing a more secure workout in individuals with cardiovascular risk.

Set configuration also affects post-exercise blood pressure. In the Failure session, both systolic and diastolic blood pressures were reduced with respect to the Control session. However, after Inter-repetition rest session blood pressure remained unaffected and not differences were found in comparison with the control session. A possible explanation to this difference is a local post-exercise vasodilation in the active muscles after Failure session due to an activation of histamine H_1 and H_2 receptors (147) as a consequence of the metabolic production associated with muscular fatigue. Postexercise hypotension has previously been observed in protocols leading to failure (142,145), even with low volume (142), what suggests that muscle failure is a substantial contributor to the onset of hypotensive effect. A previous study (11) compared a muscle failure with muscle non-failure protocols, observing a hypotensive effect only in the former. Unfortunately, in that study the load was not equated.

To the best of our knowledge, the current is the first study that compares a failure session with a protocol without failure maintaining equated all the parameters of the load. The type of exercise did not affect the blood pressure recovery and did not lead to postexercise hypotension. This lack of different between exercises is coincident with a previous study (187) that evaluated the effect of muscle mass on hypotension post-exercise using a similar volume to the present experiment. In addition, the significant DBP elevation observed in Control session could be attributed to the orthostatic stress (269). The orthostatic stress is commonly observed in prolonged sitting, possible due to a baroreflex-mediated raise in total peripheral resistance (269).

Taking this into account, long set protocols leading to failure may have practical applications as a non-pharmacological therapy in hypertensive individuals to reduce blood pressure, since muscular failure *per se*, with all the rest loading parameters equated between protocols, appears to have an important role in the onset on postexercise hypotension.

Our study in healthy young population makes a first attempt to explore the possible implications of exercise type and set configuration on participants at cardiovascular risk. Future studies should focus on analyzing the applicability of these findings on diseased individuals.

4.5. Conclusions

In summary, the type of exercise affects the cardiac vagal autonomic control after a resistance exercise, with higher reductions in Parallel squat in comparison with Bench press. Also, our data showed that Failure session caused a loss of cardiac vagal control meanwhile Inter-repetition rest session blunted the impact of resistance exercise on the post-exercise cardiac vagal control. Interactions between the type of exercise and set configurations showed that the cardiac vagal control after resistance exercise is affected by both factors simultaneously. Finally, postexercise hypotension was dependent on set configuration, with lower values of blood pressure after Failure session but not after Inter-repetition rest session.

The type of exercise and the set configuration should be carefully selected to prescribe to populations with cardiovascular risk. Exercises with a lying position and less muscle mass involved (i.e. bench press) in combination with shorter set configurations (i.e. inter-repetition rest design) could blunt the increased cardiac risk associated to a decreased vagal activity after resistance exercise. On the contrary, when the objective of the prescribed resistance exercise is a postexercise reduction in blood pressure, set configurations to failure may be recommendable. It is important to note that one simple exercise with a long set configuration was sufficient to reduce the cardiac vagal control and to provoke the onset of the postexercise hypotension in comparison to a control session.

5. Study III

Effects of set configuration of resistance exercise on perceived exertion

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Abstract

Set configuration refers to the repetitions performed with regard to the maximum possible number of repetitions, a factor affecting RPE that has not been previously studied. This study analyzed the effect of set configuration and muscle mass on RPE. Eight students (M age = 23.7 yr. ± 1.7) completed four sessions corresponding to types of exercise with different amount of muscle mass (bench press and parallel squat) and two set configurations: a session with five sets of repetitions to failure and a cluster session. The cluster session involved the same intensity, volume, and rest than the failure session, guaranteeing the same work-to-rest ratio. RPE was higher in Failure vs Cluster sessions and higher in parallel squat vs bench press. This suggests that set configuration influences RPE. Similarly, RPE can be affected by the muscle mass of the exercise performed.

5.1. Introduction

Rating of perceived exertion (RPE) scales have been used traditionally in aerobic exercise and recently, investigations have examined the usefulness of these scales to prescribe resistance exercise (35,245,246). RPE scales subjectively measure the intensity of effort, strain, discomfort and/or fatigue that a subject feels (217). Several components of resistance training have been shown to affect RPE, these include intensity (35), volume (36) and rest periods between set and exercise (232). Also, it has previously reported that RPE is related to certain physiological variables like muscle activity (246), blood lactate (245) and salivary cortisol (254).

Another factor that could influence RPE is set configuration, since it is associated with intensity, volume and metabolic response to a workload (71,270). Set configuration refers to the repetitions performed with regard to the maximum possible number of repetitions. It is an important aspect to consider when prescribing resistance training, because distribution of work and rest into subsequent sets clearly differs in the mechanical performance and metabolic responses to the exercise (71). Traditional set configuration with repetitions leading to failure causes fatigue that results in a decline in mechanical performance (73) and requires long periods of rest to sustain the number of repetition over successive sets (270).

Previous studies have shown that for traditional training with repetitions performed in a continuous fashion, total work (i.e., kg) seems to be an important factor affecting RPE (243,244). These studies compared different intensities and resistance training schemes but equated to total work, reporting similar RPE responses. However, total resting time was not the same and therefore the work-to-rest ratio was not equated.

An alternative set configuration consists of manipulating work and rest periods by breaking sets into small clusters of repetitions. This type of training has been termed cluster training, inter-repetition rest training or intra-set rest training (64,65,75). Cluster training allows a higher mechanical performance and a larger number of repetitions until failure with a lower glycolytic metabolism than the traditional training to failure (77,80). However, long-term studies have found that adaptations to cluster training vs. traditional training may be better (69), similar (70) or worse (39). The effect of a clustered set design on RPE has previously been studied (232,233). Hardee et al., (232) compared three protocols with the same intensity and total work in one protocol without inter-repetition rest, and two others with 20 sec. or 40 sec between each repetition. The study reported lower RPE values and higher mechanical performance in the 40 sec. inter-repetition rest protocol. However, work-to-rest ratio between conditions was not equated. It appears that only one study (233) equated the protocols with the same work-to-rest ratio between conditions. This study found decreased values of RPE in protocols with lower number of repetitions per set. However, a fixed number of repetitions were performed, not taking into account set configuration. It is possible that even when the intensity and work-to-rest ratio are equated, besides the total work, set configuration may affect the RPE response, as different set configurations with the same work-to-rest ratio affect to mechanical and metabolic responses to exercise (77).

Additionally, it has pointed out previously that muscle mass may affect RPE. It could be interesting to know how muscle mass affects on RPE, in order to prescribe different exercises at the same intensity (i.e., load) by perceived exertion. Exercises with large muscle mass may cause higher RPE responses. In this sense, discrepancies has been reported in the literature stating that more muscle mass may cause higher (234), the same (36), or lower (235) RPE response. Thus, it will be important to further understand the influence of the muscle mass as well as the effect of the interaction between the muscle mass and set configuration on perceived exertion during the performance of resistance exercise.

This study compared the effects of different set configurations with the same intensity (i.e., load), volume (i.e., repetitions × kg), and work-to-rest ratio on RPE and mechanical performance. Secondly, the influence of muscle mass and set configuration on perceived exertion was assessed. A protocol with sets of repetitions leading to failure (Failure session) and a cluster set configuration (Cluster session) with rest between each repetition were compared. Both protocols were performed for two types of exercise engaging different amounts of muscle mass: bench press and parallel squat. Our hypothesis is that RPE will be higher in the Failure session compared with the Cluster session, due to higher physiological demand indicated by a reduced mechanical performance and that Parallel squat will be associated with higher RPE than bench press as a result of the larger muscle mass involved.

5.2. Methods

Participants for this study were 8 healthy (7 men, 1 woman), moderately trained sport science students (*M* age= 23.8 yr. ± 1.4; *M* height= 1.7 m ± 0.1; *M* body mass= 66.75 kg ± 9.5; *M* 10RM bench press= 47.31 kg ± 15.78; *M* 10RM parallel squat= 86.00 kg ± 23.89) with at least 6 mo. of experience in resistance training lifting weights two or three times/week. All participants signed an informed consent (Appendix B) and were informed they could withdraw from the study at any time. The study was approved by the local Institutional Ethics Committee.

Procedures A repeated measures design was used in which participants completed 4 experimental trials. The experiment consisted of 9 sessions: 5 orientation sessions and 4 experimental sessions. During the course of testing, participants were instructed to refrain from exercise, alcohol, caffeine and nicotine for the 24 hr. before the testing sessions. Each session started with a warm-up of 5 min. of submaximal treadmill exercise, 5 min. of joint mobilization and calisthenics, and 2 sets of 10 repetitions with light loads.

Orientation sessions. Participants completed 3 familiarization sessions. In these sessions participants performed five progressive submaximal sets of 10 repetitions in which were given instructions on how to perform properly the exercises and learned how to anchor and rate the perceived exertion with the OMNI-RES scale following the procedures of Robertson, et al., (213). The following two sessions were performed to test the 10RM and to establish reliability. Both exercises were performed using a Smith Machine (Life Fitness, Brunswick Corporation, USA). In the bench press exercise, participants were instructed to start with the elbows fully extended and lower the bar to the chest with controlled velocity. After a pause of approximately 1 sec., participants performed the concentric phase in an explosive fashion, at maximum velocity. In the parallel squat exercise, participants started from the upright position with the knees fully extended, with the feet parallel and placed shoulder width apart and the barbell resting across the back. Participants then lowered in a controlled fashion until the thigh was in a horizontal plane, with the angle of the knee flexion at approximately 90º. Finally, participants recovered the initial position performing each repetition at the maximum velocity. All participants were provided with verbal encouragement by the same researcher. The procedures of the 10RM tests were followed as reported previously (172). 10RM was defined as the load that a participant was able to lift properly 10 times, but not 11. Participants performed no more than 5 attempts on each exercise with a rest interval of 2-5 min. between attempts. Intra-class Correlation Coefficients (ICC) were calculated to establish the reliability of the tests.

Experimental sessions. Each participant completed four sessions corresponding to two types of exercise (bench press and parallel squat) and two types of set configuration (Failure session and Cluster session). For each exercise, the components of work (i.e., intensity, volume, total rest and total weight) were equated between set configurations in order to guarantee the same work-to-rest ratio with different distribution of pauses. The order of the exercises was counterbalanced, while sessions to failure were performed prior to cluster sessions.

Work-to-rest ratio was the same in both protocols for each exercise: the Failure session consisted of 5 sets to failure with 10RM load and with 3 min. of rest between sets. The Cluster session consisted of the same number of repetitions performed in Failure session and their total rest (i.e., 4 rest periods of 3 min., 720 sec.), but with rest distributed between each repetition. Since the number of repetitions in the Cluster session depended on the volume completed in Failure session, it was not possible to randomize the order of the Failure session and Cluster session conditions. As not all participants performed the same number of repetitions, the rest intervals between repetitions during Cluster session were individualized. The number of rest intervals was equal to the number of repetitions performed minus 1 (e. g., 3 repetitions entail resting from repetition 1 to 2 and from repetitions 2 to repetition 3), pause between repetitions was calculated as [720/(numbers of repetitions completed in Failure session – 1)] sec. The mean of the length of rests between repetitions was of 24.7 sec. (*SD*= 4.4) in bench press and 21.9 sec. (*SD*= 3.5) in parallel squat. Repetitions performed in Failure session for both exercises are reported in Table 5. Participants were able to complete all repetitions in Cluster session for both exercises. Sessions were separated by at least 72 hr. and were performed by each participant at the same hour of the day.

Table 5. Repetitions performed in Failure session for Bench press and Parallel squat (n=8)

Set	Bench Press		Parallel Squat	
	M	*SD*	*M*	*SD*
Set 1	10.75	1.58	13.00	2.14
Set 2	6.88	0.83	7.50	2.73
Set 3	5.25	1.16	6.00	2.33
Set 4	4.25	1.49	4.13	2.80
Set 5	3.75	1.28	4.13	2.53
Session	30.87	5.08	34.75	6.06

Data are displayed as means±SD

Measurement of dynamic performance. A dynamic measurement device (T-Force System, Ergotech, Spain) was employed for evaluating mechanical performance. The velocities reported correspond to mean propulsive velocity (MPV) of the concentric phase of each repetition. The propulsive phase was the portion of the concentric phase during which acceleration of the barbell was greater than acceleration due to gravity.

Assessment of perceived exertion. OMNI-RES scale was used to monitor perceived exertion. The OMNI-RES was used because it has been developed specifically for resistance exercise. This scale has pictorial descriptors that can help numerical and verbal descriptors to anchor the RPE (238). Concurrent validity (213) and construct validity (218) of OMNI-RES have been studied in resistance exercise suggesting that OMNI-RES is an appropriate and valid tool for assessing perceived exertion. The instructions used followed the guidelines published elsewhere (213). The scale was anchored using exercise-memory anchoring (35,216). In order to establish anchoring, the Low anchoring was fixed performing one repetition without load, having the equivalent of 0; the High anchoring was fixed thinking in the maximum effort that participants ever performed, the equivalent of 10 (271). Ratings of perceived exertion specific to the active muscles (218) were used. Active muscles RPE reflects the peripheral perceptual signals of the exercise done. Participants were told to focus on *pectoris major* muscle in bench press, and on the *gluteus maximus*, *quadriceps* and hamstrings in parallel squat. The same trained researcher explained and asked RPE values in the orientation and experimental sessions. OMNI-RES scale was in full view of the participants at all times during the procedures. In the Failure session, OMNI-RES was taken at the end of each set. Participants reported immediately a number of the OMNI-RES (0–10) scale decided simultaneously with the repetition of the set in which the participant failed to lift the weight. In the Cluster session, OMNI-RES value was reported immediately after the concentric phase of the repetition number coincident with the end of each set in the Failure session.

Analysis. Descriptive statistics are shown as mean±*SD*. Reliability of 10RM load was assessed by Intra-Class Correlation Coefficient (ICC) calculated using the one-way random effect model. Only Single Measure Intra-Class correlation was considered. A 2-way repeated measures ANOVA (exercise × set) was performed to compare the number of repetitions performed across the 5 sets in Failure sessions. Three-way repeated measures analyses of variance (ANOVA) was used to evaluate the effect of session (Failure or Cluster), exercise (bench press or parallel squat) and time (Set 1, Set 2, Set 3, Set 4, Set 5) on RPE. Also, a two-way repeated measures ANOVA (session × exercise) was performed for Set 5 on RPE in order to compare set configurations and exercises when accumulated resting time was the same between conditions. Additionally, a two-way repeated measures ANOVA (session × time) was performed for both exercises to identity any differences in MPV. When necessary, multiple comparisons were performed with Bonferroni correction. Statistical significance was assumed at $p \leq .05$. Effect sizes are reported as omega squared (ω^2). The data were analyzed using SPSS Version 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-β) of a repeated-measures ANOVA with 5 measurements for a sample size of 8 and a large effect size (f= 0.4) is 0.754.

5.3. Results

Reliability of the 10RM across 2 trials was high for both exercises (ICC= .99 and .95 for bench press and parallel squat, respectively). Two-way repeated measures ANOVA indicated that there were no differences in the number of repetitions between exercises ($F_{1,7}$= 2.67, p= .15), nor an interaction between exercise and sets ($F_{4,28}$= 1.07, p= .39). A significant effect of set was detected, showing a progressive decrease in the number of repetitions with each successive set ($F_{4,28}$= 42.93, p< .001).

Table 6 shows the values of OMNI-RES and MPV for every session, exercise, and set in Failure session or the coincident repetition in Cluster session. Table 7 summarizes main effects, effect sizes, and interactions of the OMNI-RES responses.

The results of the 3-way ANOVA indicated significant main effects of session, exercise, and sets. The main effect for session indicated that RPEs were higher in the Failure session than the Cluster session for both exercises ($F_{1,7}$= 12.47, *p*= .01). Furthermore, the main effect for exercise showed that rates of perceived exertion in parallel squat were higher compared with bench press ($F_{1,7}$= 23.73, *p*= .002). The main effect for sets indicated an increase of RPE with each subsequent set ($F_{1,7}$= 13.81, *p*= .002). A significant interaction was observed for Session × Exercise ($F_{1,7}$= 11.71, *p*= .01). Simple effects indicated that RPE was lower during the Cluster session than the Failure session for bench press ($F_{1,7}$= 17.79, *p*= .004), but not for parallel squat. Also, differences between exercises were only observed for the Cluster session, with higher values in parallel squat than bench press ($F_{1,7}$= 27.32, *p*< .001).

Two-way ANOVA of data corresponding to the final repetition performed in both protocols, when total resting time was equated between conditions, showed significant differences between sessions (Failure session= 9.38±1.03; Cluster session= 7.81±1.61; $F_{1,7}$= 7.72, *p*= .03) and exercises (bench press= 8.19±1.61; parallel squat= 9±1.38; $F_{1,7}$ = 18.78, *p*= .003). Similarly, the interaction between session and exercise was significant ($F_{1,7=}$ 38.16, *p*< .001).

Analyses of simple effects indicated that RPEs were lower during the Cluster session than the Failure session for bench press ($F_{1,7=}$ 14.44, *p*= .007), but not for parallel squat. In addition, differences between exercises were significant only during the Cluster session, with higher values in parallel squat with respect to bench press ($F_{1,7=}$ 38.16, *p*< .001).

Table 6. OMNI-RES responses and MPV through sessions, exercises and sets (n=8)

Set	Failure Session				Cluster Session			
	OMNI		MPV		OMNI		MPV	
	M	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
				Bench press				
Set 1	7.38	1.47	.321	.069	5.16	1.67	.388	.051
Set 2	8.00	1.13	.258	.049	5.75	1.51	.370	.053
Set 3	8.75	2.4	.240	.054	6.00	1.4	.353	.058
Set 4	9.13	2.43	.235	.072	6.62	1.18	.341	.074
Set 5	9.38	2.18	.229	.068	7.00	1.30	.314	.071
Mean	8.53	1.52	.272	.062	6.10	1.53	.364	.055
				Parallel Squat				
Set 1	8.25	1.89	.335	.043	6.50	1.69	.368	.044
Set 2	8.87	1.35	.321	.035	7.50	1.69	.341	.051
Set 3	9.12	.99	.303	.037	7.87	1.35	.337	.055
Set 4	8.75	1.48	.309	.042	8.25	1.38	.322	.040
Set 5	9.37	1.18	.299	.043	8.62	1.30	.308	.078
Mean	8.88	1.39	.321	.034	7.75	1.6	.346	.048

Data are displayed as means±SD

For MPV, there were significant differences in session for bench press, with higher values in the Cluster session than the Failure session ($F_{1,7=}$ 17.49, p= .004). However, there were no differences between sessions in parallel squat ($F_{1,7=}$ 1.28, p= .29). A time effect was observed for both bench press ($F_{4,28=}$ 32.93, p< .001) and parallel squat ($F_{4,28=}$ 8.29, p< .001), with lower values in each subsequent set (Table 7).

Table 7. OMNI-RES values for main effects and interactions (n=8)

Source		*M*	*SD*	p	ω^2
Session	Failure	8.70	1.45	.01	
	Cluster	6.93	1.76		.23
Exercise	Bench press	7.31	1.78	.002	
	Parallel squat	8.31	1.35		.070
Sets	Set 1	6.81	2.07	.002	
	Set 2	7.53†	1.87		
	Set 3	7.94†	1.72		
	Set 4	8.19	1.57		
	Set 5	8.59†‡	1.50		.29
Session x Exercise				.01	.03
Session x Sets				.33	.00
Exercise x Sets				.1	.00
Session x Exercise x Sets				.063	.00

Data are means±SD †Different from Set 1 (p <.05) ‡Different from Set 2 (p <.05)

5.4. Discussion

The main findings of this study were that the Failure session induced higher RPE compared with the Cluster session; when comparing the types of exercise, RPEs for parallel squat were higher than bench press; and set configuration affected both RPE and MPV in the same way. The results showed that when intensity, volume, and work-to-rest ratio were equated, the Cluster session lead to lower RPE. Hence, the set configuration seems to influence perceived exertion responses. A previous study (233) analyzed different set configurations with equated work-to-rest ratios, obtaining similar results and showing lower RPEs for the set configuration with fewer repetitions per set. However, this study (233) employed a fixed number of repetitions at an established intensity (i.e., 60% of 1 RM), so the ratio between repetitions performed and the maximum number of repetitions was not taken into account.

In the current study, the load was set according to the maximum number of repetitions that could be completed (i.e., 10RM load). During the Failure session, participants performed 5 sets to failure with the 10RM load, which entailed a decrease in the number of repetitions in successive sets in a similar way to data previously reported (270). In the Cluster session, participants performed the same number of repetitions as the Failure session, but with total rest distributed between repetitions. Therefore, this study design allows the comparison of different set configurations with the rest of the load parameters equated.

Some studies found no differences in protocols with the same total work (243,244), concluding that total weight lifted seems to be an important factor affecting RPE in traditional protocols. Nevertheless, they used protocols with different intensities and the total resting time between protocols was not the same, therefore the work-to-rest ratio was not equated. Another study (232) compared three protocols with the same intensity and total work: a protocol without inter-repetition rest and other two with 20 sec. or 40 sec. between each repetition.

The study reported lower RPE values with the 40 sec. inter-repetition rest protocol in comparison with 20 sec. and the traditional protocol. However, work-to-rest ratio between conditions was not equated. In the current study, with the same intensity, total weight lifted, and work-to-rest ratio equated, differences were observed between protocols. This suggests that not only total weight lifted, but also set configuration affects RPE response.

Pairwise comparisons of RPE revealed that differences between sessions were only observed for bench press. These results are consistent with mechanical performance values since differences in MPV were only observed for this exercise. For bench press, mechanical performance was higher in the Cluster session in comparison with the Failure session, in contrast to the RPE response reported in the literature previously (232). In the parallel squat, no differences were observed for RPE or MPV. This suggests that RPE response represents properly the mechanical performance differences of the sessions.

Differences between exercises revealed higher RPEs for the parallel squat than in the bench press, suggesting that muscle mass may affect the RPE response. Higher RPEs to exercises involving larger muscle mass have been previously reported (234). However, others have not observed differences between exercises (36) or have found lower RPE for the exercises recruiting more muscle mass, in a sample of children (235). In addition to this, same intensity with regard to the maximum possible number of repetitions (i.e., 10RM) can mean different intensities respect to the percentage of 1RM, so it is conceivable that the exercise with large muscle mass is in a higher percentage of 1RM (49), so a higher RPE response is expected (35). This is in agreement with the hypothesis that at same RM load, exercises recruiting more muscle mass could result in higher RPEs. At least with the load of a goal RM, exercises requiring different amounts of muscle mass may evoke different RPEs. Nevertheless, as studies were not randomized (36,234) or were conducted in children (235), further studies should be conducted to identify the role of the muscle mass on RPE.

Additionally, analysis of interaction only detected differences between exercises in the Cluster session. RPEs in the Failure session suggest a ceiling effect, which is inherent to category-ratio scales when the perception of fatigue is extremely high (217,223). This limitation has been reported in other studies with protocols to failure (222,272), suggesting that RPE scales are more useful to assess submaximal designs that protocols that lead to failure. Overview of the data suggests that the RPE differences between exercises could be influenced by the set configuration.

5.5. Conclusions

In summary, the results demonstrate that set configuration may have an important role in RPE response, and may be influenced by the muscle mass involved. Also, RPE may have practical applications for monitoring resistance exercise, since it reflects differences between protocols with different set configurations and represents properly the mechanical performance of the exercises. The results provide evidence that perceived exertion values in similar designs (i.e., same intensity and work-to-rest ratio) at submaximal protocols are influenced by the muscle mass, suggesting that a fixed value of RPE does not represent the same intensity in different exercises, so attention should be paid when prescribing the intensity using RPE for different exercises.

6. General discussion

The main findings of this thesis were that: a) maximal and submaximal long set configurations produce a higher reduction in cardiac parasympathetic activity as observed by the loss in autonomic and baroreflex control in comparison with short set configurations. b) a short set configuration as an inter-repetition rest design preserves the parasympathetic control of the heart as observed by non-significant reductions of autonomic and baroreflex control after resistance exercise. c) the effect of the set configuration on vagal autonomic control is affected by the type of exercise performed during the inter-rest repetition design, with a higher preservation of parasympathetic activity in the exercise with less muscle mass involved, in comparison with the exercise with more muscule mass involved; but not during a maximal set configuration. d) maximal but not submaximal long set configurations produce the onset of post-exercise hypotension in a session with a small total volume. e) the effect of the set configuration on post-exercise hypotension is not modulated by the type of exercise in a session with a small total volume. f) a maximal set produces a higher perceived exertion in comparison with a short set configuration as an inter-repetition rest design, indicating that the set configuration determines the perceived exertion. g) the effect of the set configuration on perceived exertion was dependent on the exercise performed, with differences observed between protocols in the exercise with less mucle mass involved but not between protocolos with more muscle mass involved.

The results of these studies showed that when the intensity of load, volume and work-to-rest ratio were equated, the set configuration used determines the loss of parasympathetic activity as observed by the loss in autonomic and baroreflex control, in disregard of the comparison between submaximal protocols or between submaximal versus maximal designs.

Thus, the affectation of the set configuration on the parasympathetic control is independent of the appearance or not of muscle failure in the comparison between protocols.

A major candidate for the loss of vagal autonomic control of the heart after exercise is the glycolytic involvement of the session, with higher reductions in parasympathetic activity with higher lactate productions (103,107,108,268). These relationships between cardiac parasympathetic control and glycolytic involvement were observed previously after intravenous injections of lactate (108,268) and resistance exercise sessions (103,107). Although lactate production was not analyzed in our study, former studies have shown differences in the glycolytic involvement between long and short set configurations after a resistance exercise session (66,71,82–84). In addition, lactate production correlates strongly with mechanical performance (71), and the short set configuration protocols in these studies showed a higher manifestation of velocity during the session than the long set configurations. Those arguments support the inference that the differences between set configurations may be mediated by the glycolytic involvement of the session.

The present results for the longer set configurations support previous data reporting that resistance exercise perturbs the autonomic control inducing a reduction in cardiac vagal control (4,5,11–13). Nevertheless, no significant reductions were observed after the inter-repetition rest designs, and this is a new finding. These non-significant reductions may be due to non-significant elevations of lactate production as previously observed in an inter-repetition rest design (77). The no significant elevations of lactate production occurred are probably the result of a partial regeneration of PCr that allows a lower demand of anaerobic glycolysis (66).

When taking into account the exercise performed in the inter-repetition rest design, no significant reductions in cardiac vagal autonomic activity were observed when performing leg press (Study I) and bench press (Study II), but a significant decrease comparable to the long set configuration was observed when performing parallel squat (Study II).

Peculiarly, in agreement with our study, a previous research using parallel squat to analyze the effect of set configuration on the cardiac autonomic control reported no differences between protocols (102). While drawing inferences about the reasons for these differences would be nowadays misleading, appears that the ability of short set configurations as the inter-repetition rest design to reduce the loss of cardiac vagal control after resistance exercise may be exercise-dependant.

Regarding performing the same set configuration with different types of exercise, our data showed differences between exercises while performing short set configurations. In this sense, lactate production may account for the differences between bench press and parallel squat as was previously observed (71). Nevertheless, protocols leading to muscular failure but differing in the type of exercise had the same cardiac vagal activity after exercise. This is an interesting point because it is known that bench press and parallel squat produce different amounts of lactate during consecutive sets to failure (71). Similar to our results, a previous study observed no differences comparing protocols with exercises of upper limbs versus lower limbs leading to muscular failure (13). These findings suggest that other physiological factors that occur during muscular failure should displace the importance of the effect of lactate production, having the former a preferential role in the reduction of cardiac vagal control.

Cardiac parasympathetic baroreflex control was also affected by the set configuration, with higher reductions in baroreflex sensitivity with longer set configurations in comparison with the inter-repetition rest design (Study I: 8S and 4S vs. 1S). Our results support previous observations showing that a resistance exercise session causes a transient reduction in the cardiac parasympathetic baroreflex control (14–16). While all the previous studies were performed with several exercises and showed significant reductions in baroreflex sensitivity (14–16), this is the first design to present that a single resistance exercise is enough to affect the baroreflex control.

Also, our study shows that with short set configurations as an inter-repetition rest design is possible to perform resistance exercise without reductions in baroreflex sensitivity. Additionally, our results support a previous study showing that more demanding protocols cause a higher decrease in baroreflex sensitivity than less demanding protocols (15). Differences in physiological demands can be interpreted as differences in the mechanical performance, a proxy of neuromuscular fatigue (71). While the factors affecting cardiac baroreflex control are not elucidated yet, it is possible that increases in nitric oxide (131) and arterial stiffness (14) partially explain the reduction of baroreflex sensitivity. On the one hand, increases in nitric oxide as a consequence of an augmented shear stress during resistance exercise (206) may reduce the sensitivity of the baroreflex, as was previously observed with nitric oxide injection (131). On the other hand, an increased arterial stiffness in the central vessels after exercise as a consequence of increased reservoir pressure may reduce the wall deformation and thus attenuate the baroreceptor activation (14).

The control of the reduction in cardiac parasympathetic activity after resistance exercise may have practical applications in individuals with cardiac risk. This is because the reduction in cardiac parasympathetic control after exercise can be interpreted as a transient deleterious effect, since 30 min after an exercise there is a increased risk of a sudden cardiac death to decreased vagal activity (17). In addition, prognosis studies showed that decrements in the parasympathetic branch of the autonomic and baroreflex control are associated with myocardiac ischemia and cardiac death (97), suggesting the pertinence of maintaining the maximal parasympathetic activity possible after exercise in individuals with certain cardiac risk. This is due to the role that the parasympathetic activity has a regulator of the ventricular electrophysiological properties, providing protection against ventricular arrhythmias (273,274).

Beyond administrating the loss of cardiac parasympathetic control, our studies also provide data that can be possible to perform several exercises of resistance training (i.e., bench press and leg press) without eliciting the cardiac impact that causes a reduction in autonomic and baroreflex activity: This has clear relevance in the prescription of exercise in diseased individuals. Future studies should analyze if the theoretical benefits of a non-significant reduction with an exercise are consistent in a whole resistance training session. Besides, it is of interest the effect that different set configurations on parasympathetic control may have after a resistance training period in individuals with an affected autonomic control, and if dissimilar configurations of the set produce or not improvements in the vagal control. In this sense, it is known that in populations with an altered autonomic control, long set configurations may improve vagal activity (10), but the effect of short sets are still unknown.

Set configuration also determines post-exercise hypotension. Our results revealed that when the intensity of load, volume, and work-to-rest ratio were equated, a maximal set configuration, with an intensity of effort of 100%, produces a reduction in systolic and diastolic blood pressure in comparison with a control session (Study II: Failure session). On the contrary, submaximal long (Study I: 8S) and short set configurations (Study I: 1S, and Study II: Inter-repetition rest session) did not produce post-exercise hypotension. These findings support previous studies that reported that reaching muscular failure was a co-factor of the onset on post-exercise hypotension (11,172,196). Nevertheless, those studies were performed with different intensities of load, which can affect the results (11,172,196). To our knowledge, our study (Study II) is the first that compared a protocol leading to failure with another without muscular failure, with the rest of the loading parameters remaining equated. In this regard, attending to our results, long set configurations without leading to failure (Study II: 8S) and short set configurations (Study I: 4S and 1S. Study II: Inter-repetition rest session) do not cause the onset of post-exercise hypotension with a kind of protocol that may do that, indicating that those types of sets could not be the better designs when post-exercise hypotension is desired.

Additionally, in our study, the interaction between the set configuration and the type of exercise reported no differences. Regarding the short sets, no effects between inter-repetition rest designs with different exercises are expected, as these types of design do not cause post-exercise hypotension. Regarding the sets leading to failure, this is consistent with a previous study analyzing upper versus lower limbs with a comparable volume (6). It appears that to benefit from the exercise type when comparing upper and lower limbs, the volume should be higher than ours (146). In this sense, as previously explained, seems that in our studies muscular failure were the major determinant of post-exercise hypotension.

Previous studies have reported that a minimum volume is necessary, even when reaching muscular failure, for the onset of post-exercise hypotension after resistance exercise (6,15). On this point, data from a previous study suggested that the total volume performed is crucial for post-exercise hypotension, exceeding the effect of muscular failure (172). Our study, with a progressive reduction in the number of repetitions in each set due to fatigue, was sufficient to reveal a post-exercise hypotension with a very low total volume (about 32-34 total repetitions). This challenges in part the standpoint that the total volume is the cornerstone loading parameter that determines post-exercise hypotension after resistance exercise, since until date the design with the lower volume that produced hypotensive effect was about 100 repetitions (6). Nevertheless, this finding is not contradictory with the role of total volume performed, since the physiological founder for both features of the session may be the same.

In this regard, of the obligatory mechanisms for the onset of post-exercise hypotension, the vasodilatation dependent of the activation of the H_1 (156) and H_2 (157) receptors seems to be the factor that occurs during muscular failure, and probably while performing large volumes.

This is because during muscular failure there are large muscular contractions, an affected blood flow, and the inability to perform new repetitions due to fatigue. All of those features are contributors to the release or production of histamine or contributors to the activation of histamine receptors (203–205), since the activation of histamine receptors protects against fatigue during exercise (152) and modulates the metabolism during recovery (207). The same can be argued for the effect of total volume performed on post-exercise hypotension, since during every new set larger muscular contractions, worsened blood flow, and progressive muscle fatigue are expected to occur. Our findings defy the previous data reported, since muscular failure was the prime intended effect in our study, as observed by the progressive reduction in the number of repetitions performed in each set due to an incapacitating fatigue. Thus, intentional and deliberate muscular failure may displace the effect of the total volume performed to a secondary role.

The reduction in post-exercise blood pressure after the Failure session design (Study II) has a practical application in training as a non-pharmacological strategy to treat or prevent the appearance of hypertension (139). This is due that post-exercise hypotension may contribute to the chronic reductions in blood pressure (140) as observed by the correlations between post-exercise hypotension and long-term blood pressure reduction after resistance training (141). Notwithstanding, at the same time that maximal set configurations lead to post-exercise hypotension, they also lead to a large decrease in cardiac parasympathetic control, as was previously indicated. The suitability of this kind of training should be dependent on the cardiac health that the individual has as measured by the grade of autonomic activity and by the pertinence of the session as inspected by the characteristics of the person (100). Future studies should focus on the potentiality of maximal long set configurations in the reduction of post-exercise blood pressure with a whole resistance training session, and on their possible relation with the activation of histamine receptors as was previously analyzed in aerobic exercise (156,157). In this sense, just one previous study analyzed the effect of resistance exercise, specifically isokinetic, on post-exercise blood pressure, without leading to failure and without finding post-exercise hypotension (152).

Lastly, the set configuration also determines perceived exertion. Our results indicated, for the first time, that when the intensity of load, the volume, and the ratio between work and rest are equated, a maximal set configuration (Study III: Failure session) produces a higher perceived exertion than a short set configuration as an inter-repetition rest design (Study III: Cluster session). Our data is coincident with a previous study that compared protocols with the same intensity of load, volume and work-to-rest ratio but that used a percentage of the 1RM, not taking into account the maximum number of repetitions that can be performed (233). That was a weakness in that design since the number of repetitions is individual-dependant (45–50) and the remoteness and closeness to muscular failure of each individual is a confounding factor in relation to perceived exertion (237,242–244).

Our findings support previous research that argued that the duration of the repetitions (255) and the length of the set (213) mediate perceived exertion. Then, the appearance of failure is a great determinant of perceived exertion (236,237) since the repetitions and the length of the set are obviously largest with each longer configuration of the set. On this point, when comparing maximal versus submaximal set configurations, two reasonings should be unfolded. On the one hand, previous experiments altering the work-to-rest ratio observed that the lower work-to-rest ratio caused lower perceived exertion values, explaining the differences between protocols (37,233). Nevertheless, in our study, matching the work-to-rest ratio, differences between our protocols still exist, showing a secondary role for that ratio between work and rest when maximal and submaximal set configurations are compared. On the other hand, with the intensity of load (237) and volume (231) equated, the response of perceived exertion should be similar between protocols, as was previously debated.

Nevertheless, when comparing a maximal versus submaximal set configurations, intensity of load and volume seem to be a minor role affecting perceived exertion. Taking all this into account, it can be argued that the dissimilarities in our study are mediated by the differences in the duration of the repetitions (255) and the length of the sets (213), as observed by the remoteness and closeness to the muscular failure, which is a determinant of perceived exertion (236,237).

In regards to the interaction between the set configuration and the type of exercise, we observed that in the type of exercise with less muscle mass involved (Study III: Bench press), set configuration caused different values of perceived exertion, with higher values in the maximal set configuration (Study III: Failure session). Nevertheless, in the exercise with more muscle mass involved (Study III: Parallel squat), no differences were observed in perceived exertion between sets configurations, despite higher values in the Failure session. The physiological reasons for those differences are unknown. Nevertheless our results mirror the differences of mechanical performances between set configurations for the same exercise, as differences were reported for Bench press but not for Parallel squat. While physiological determinants of perceived exertion are not clarified, a mirroring between perceived exertion and mechanical performance is expected, as observed previously between perceived exertion and lactate production (213,248–251) and muscle activity (245,246), both proxies of neuromuscular fatigue.

In the same way, differences between exercises were only observed for the short set configuration (Study III: Cluster session), but not between maximal set configurations. In the Cluster session, higher values were observed for Parallel squat, the exercise with more muscle mass involved, which is concordant with a previous study that argued that more muscle mass involved produces more perceived exertion (234). While differences in lactate production (71) may account in part for those differences between protocols, as perceived exertion and lactate production behave in a corresponding manner (213,248–251), a reasonable justification for this difference is the higher relative intensity of load for Parallel squat in comparison with Bench press (49), which could affect perceived exertion (35,218,224).

Controling perceived exertion through set configuration may have practical applications of interest during a process of training. In this sense, set configuration can help to regulate and modulate the perceptive responses that affect an individual during resistance training. This may detect and prevent negative processes that lead to illness and overtraining (33) with the help of other parameters of the session. Otherwise, short set configurations as the inter-repetition design may help to adhere the participants to a resistance training program, since lower values of perceived exertion were observed to correlate with higher adherence to training (33). This is a very interesting point, since as was previously showed, the strength benefits between long and short set configurations as our inter-repetition rest design are comparable (93). Further studies are needed to analyze the effect of longer but submaximal set configurations in comparison with other short set configurations. In this sense, if they produce different perceived exertion, the prescription of strength training through set configuration may be of interest to modulate the perceptual responses during exercise.

The studies included in this thesis present several limitations that should be taken into account. Our participants, young adults without diseases, represent a good model to try to understand how resistance exercise affects cardiovascular control in healthy individuals, and how to affect the cardiac control in diseased individuals with an intact nervous system. Nevertheless, studies about the effect and applicability of set configuration are needed in diseased individuals with an affected autonomic and baroreflex control and with hypertension. In Study II, men and women were studied together, and there could be differences related to the gender of the participants. The inclusion of some metabolic markers, such as lactate production, could have provided valuable information about the autonomic and perceptive responses to resistance exercise. Additionally, the posture of the participants in the data collection (Study I: semirecumbent, Study II: seated) may account for some differences in data interpretation between protocols and in comparison with the literature about cardiovascular responses to exercise. The same can be argued with the breathing frequency and tidal volume regarding the autonomic analysis, since both are variables that can affect the interpretability of the data.

7. General conclusions

- A resistance exercise with longer set configurations produces a higher cardiac vagal withdrawal and loss in baroreflex sensitivity in comparison with shorter set configurations.

- A short set configuration as an inter-repetition rest design produces a non-significant reduction in parasympathetic activity, explained by comparable values of cardiac vagal control and baroreflex sensitivity.

- The effect of the set configuration is modulated by the type of exercise performed, with higher reductions in cardiac vagal control in long set configurations in comparison with short set configurations performing the bench press, but not performing the parallel squat.

- The effect of the type of exercise performed is modulated by the set configuration used, with a higher reduction of cardiac vagal control in the exercise with more muscle mass involved (i.e., parallel squat) in comparison with the exercise with less muscle mass involved (i.e., bench press) performing a short set configuration, but not performing sets to failure.

- Differently to the leg press and the bench press, an inter-repetition rest design performing the parallel squat produces reductions in cardiac control, suggesting that the applicability of the short set configuration designs is dependent on the type of exercise performed.

- A single resistance exercise is enough to produce a reduction in cardiac vagal activity and baroreflex sensitivity after the session.

- A resistance exercise with a short volume performed with submaximal, long or short set configurations does not produce post-exercise hypotension.

- A resistance exercise performed with a short volume but with a maximal, long set configuration produces post-exercise hypotension.

- A resistance exercise performed with a short volume produced a comparable post-exercise hypotension in disregard of the type of exercise performed.

- A resistance exercise with a long set configuration produces a higher perceived exertion in comparison with a short set configuration.

- The effect of set configuration used is modulated by the type of exercise performed, with higher values of perceived exertion in a long set configuration in comparison with a short set configuration performing the bench press, but not performing the parallel squat.

- The effect of the type of exercise performed is modulated by the set configuration used, with higher values of perceived exertion in the exercise with more muscle mass involved (i.e., parallel squat), in comparison with the exercise with less muscle mass involved (i.e., bench press) performing a short set configuration, but not performing sets to failure.

8. Bibliography

1. Hurley BF, Roth SM. Strength training in the elderly: effects on risk factors for age-related diseases. Sport Med. 2000 Oct;30(4):249–68.

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.

3. Ratamess NA, Alvar BA, Evetoch TK, Housh T, Kibler W, Kraemer WJ, et al. American College of Sports Medicine position stand. Progression models in resistance training for healthy adults. Med Sci Sports Exerc. 2009 Mar;41(3):687–708.

4. Rezk CC, Marrache RCB, Tinucci T, Mion D, Forjaz CLM. Post-resistance exercise hypotension, hemodynamics, and heart rate variability: influence of exercise intensity. Eur J Appl Physiol. 2006 Sep;98(1):105–12.

5. Queiroz ACC, Kanegusuku H, Chehuen MR, Costa LAR, Wallerstein LF, Dias da Silva VJ, et al. Cardiac work remains high after strength exercise in elderly. Int J Sports Med. 2013 May;34(5):391–7.

6. Polito MD, Farinatti PTV. The effects of muscle mass and number of sets during resistance exercise on postexercise hypotension. J Strength Cond Res. 2009 Nov;23(8):2351–7.

7. Kingsley J, Panton LB, McMillan V, Figueroa A. Cardiovascular autonomic modulation after acute resistance exercise in women with fibromyalgia. Arch Phys Med Rehabil. Elsevier Inc.; 2009 Sep;90(9):1628–34.

8. Harris KA, Holly RG. Physiological response to circuit weight training in borderline hypertensive subjects. Med Sci Sports Exerc. 1987 Jun;19(3):246–52.

9. Mota MR, de Oliveira RJ, Dutra MT, Pardono E, Terra DF, Lima RM, et al. Acute and chronic effects of resistive exercise on blood pressure in hypertensive elderly women. J Strength Cond Res. 2013 Dec;27(12):3475–80.

10. Figueroa A, Kingsley J, McMillan V, Panton LB. Resistance exercise training improves heart rate variability in women with fibromyalgia. Clin Physiol Funct Imaging. 2008 Jan;28(1):49–54.

11. De Souza JC, Tibana RA, Cavaglieri CR, Vieira DCL, De Sousa NMF, Mendes FADS, et al. Resistance exercise leading to failure versus not to failure: effects on cardiovascular control. BMC Cardiovasc Disord. 2013 Nov 19;13(1):105.

12. Heffernan KS, Sosnoff JJ, Jae SY, Gates GJ, Fernhall B. Acute resistance exercise reduces heart rate complexity and increases QTc interval. Int J Sports Med. 2008 Apr;29(4):289–93.

13. Kingsley J, Hochgesang S, Brewer A, Buxton E, Martinson M, Heidner G. Autonomic modulation in resistance-trained individuals after acute resistance exercise. Int J Sports Med. 2014 Sep;35(10):851–6.

14. Heffernan KS, Collier SR, Kelly EE, Jae SY, Fernhall B. Arterial stiffness and baroreflex sensitivity following bouts of aerobic and resistance exercise. Int J Sports Med. 2007 Mar;28(3):197–203.

15. Niemelä TH, Kiviniemi AM, Hautala AJ, Salmi JA, Linnamo V, Tulppo MP. Recovery pattern of baroreflex sensitivity after exercise. Med Sci Sport Exerc. 2008 May;40(5):864–70.

16. Queiroz ACC, Sousa JCS, Cavalli AAP, Silva ND, Costa LAR, Tobaldini E, et al. Post-resistance exercise hemodynamic and autonomic responses: Comparison between normotensive and hypertensive men. Scand J Med Sci Sports. 2014 Jul 1;1–9.

17. Albert CM, Mittleman MA, Chae CU, Lee IM, Hennekens CH, Manson JE. Triggering of sudden death from cardiac causes by vigorous exertion. N Engl J Med. 2000;343:1355–61.

18. Stanley J, Peake JM, Buchheit M. Cardiac parasympathetic reactivation following exercise: implications for training prescription. Sport Med. 2013 Dec;43(12):1259–77.

19. Kiviniemi AM, Hautala AJ, Kinnunen H, Tulppo MP. Endurance training guided individually by daily heart rate variability measurements. Eur J Appl Physiol. 2007;101(6):743–51.

20. Gomes Anunciação P, Doederlein Polito M. A review on post-exercise hypotension in hypertensive individuals. Arq Bras Cardiol. 2011 May;96(5):e100–9.

21. Cardoso CG, Gomides RS, Queiroz ACC, Pinto LG, da Silveira Lobo F, Tinucci T, et al. Acute and chronic effects of aerobic and resistance exercise on ambulatory blood pressure. Clinics (Sao Paulo). 2010 Mar;65(3):317–25.

22. Polito MD, Farinatti PTV. Blood pressure behavior after counter-resistance exercises: a systematic review on determining variables and possible mechanisms. Rev Bras Med do Esporte. 2006;12(6):386–92.

23. Cornelissen VA, Fagard RH, Coeckelberghs E, Vanhees L. Impact of resistance training on blood pressure and other cardiovascular risk factors: a meta-analysis of randomized, controlled trials. Hypertension. 2011 Nov;58(5):950–8.

24. Kelley GA, Kelley KS. Progressive resistance exercise and resting blood pressure : A meta-analysis of randomized controlled trials. Hypertension. 2000 Mar;35(3):838–43.

25. Cornelissen VA, Fagard RH. Effect of resistance training on resting blood pressure: a meta-analysis of randomized controlled trials. J Hypertens. 2005 Feb;23(2):251–9.

26. Cornelissen VA, Smart NA. Exercise training for blood pressure: a systematic review and meta-analysis. J Am Heart Assoc. 2013 Feb;2(1):e004473.

27. Kingsley JD, Figueroa A. Acute and training effects of resistance exercise on heart rate variability. Clin Physiol Funct Imaging. 2014 Dec 18 [Epub ahead of print].

28. Oliveira RS, Vitor da Costa M, Pedro RE, Polito MD, Avelar A, Cyrino ES, et al. Acute cardiac autonomic responses after a bout of resistance exercise. Sci Sports. 2012 Dec;27(6):357–64.

29. Heffernan KS, Kelly EE, Collier SR, Fernhall B. Cardiac autonomic modulation during recovery from acute endurance versus resistance exercise. Eur J Cardiovasc Prev Rehabil. 2006 Feb;13(1):80–6.

30. Figueiredo T, Rhea MR, Peterson M, Miranda H, Bentes CM, dos Reis VM de R, et al. Influence of number of sets on blood pressure and heart rate variability after a strength training session. J Strength Cond Res. 2015 Nov 26;29(6):1556–63.

31. Brito ADF, de Oliveira CVC, Santos M do SB, Santos ADC. High-intensity exercise promotes postexercise hypotension greater than moderate intensity in elderly hypertensive individuals. Clin Physiol Funct Imaging. 2014 Aug 13;34(2):126–32.

32. Borg GA. Perceived exertion: a note on “history” and methods. Med Sci Sports. 1973 Jan;5(2):90–3.

33. Foster C. Monitoring training in athletes with reference to overtraining syndrome. Med Sci Sports Exerc. 1998 Jul;30(7):1164–8.

34. Williams DM, Dunsiger S, Ciccolo JT, Lewis BA, Albrecht AE, Marcus BH. Acute affective response to a moderate-intensity exercise stimulus predicts physical activity participation 6 and 12 months later. Psychol Sport Exerc. 2008;9(3):231–45.

35. Lagally KM, Costigan EM. Anchoring procedures in reliability of ratings of perceived exertion during resistance exercise. Percept Mot Skills. 2004 Jun;98(3 Pt 2):1285–95.

36. Lins-Filho O, Robertson RJ, Farah BQ, Rodrigues SLC, Cyrino ES, Ritti-Dias RM. Effects of exercise intensity on rating of perceived exertion during a multiple-set resistance exercise session. J Strength Cond Res. 2012 Mar;26(2):466–72.

37. Farah BQ, Lima AHRA, Lins-Filho OL, Souza DJCA, Silva GQM, Robertson RJ, et al. Effects of rest interval length on rating of perceived exertion during a multiple-set resistance exercise. Percept Mot Skills. 2012 Aug 29;115(1):273–82.

38. Stone MH, Stone ME, Sands WA, Pierce KC, Newton RU, Haff GG, et al. Maximum Strength and Strength Training—A Relationship to Endurance? Strength Cond J. 2006;28(3):44.

39. Hansen KT, Cronin JB, Pickering SL, Newton MJ. Does cluster loading enhance lower body power development in preseason preparation of elite rugby union players? J Strength Cond Res. 2011 Aug;25(8):2118–26.

40. Steele J. Intensity; in-ten-si-ty; noun. 1. Often used ambiguously within resistance training. 2. Is it time to drop the term altogether? Br J Sports Med. 2014 Dec 12;48(22):1586–8.

41. González-Badillo JJ, Marques MC, Sánchez-Medina L. The importance of movement velocity as a measure to control resistance training intensity. J Hum Kinet. 2011 Sep;29A:15–9.

42. González-Badillo JJ, Sánchez-Medina L. Movement velocity as a measure of loading intensity in resistance training. Int J Sports Med. 2010 May;31(5):347–52.

43. Carpinelli R. The size principle and a critical analysis of the unsubstantiated heavier-is-better recommendation for resistance training. J Exerc Sci Fit. 2008;6(2):67–86.

44. Fisher J, Smith D. Attempting to better define “intensity” for muscular performance: is it all wasted effort? Eur J Appl Physiol. 2012 Dec;112(12):4183–5; author reply 4187–8.

45. Claiborne JM, Donolli JD. Number of repetitions at selected percentages of one repetition maximum in untrained college women. Res Q Exerc Sport. 1993;64:A39–40.

46. Hoeger WWK, Barette SL, Hale DF, Hopkins DR. Relationship between repetitions and selected percentages of one repetition maximum. J Appl Sport Sci Res. 1987 Feb;1(1):11–3.

47. Iglesias E, Boullosa DA, Dopico X, Carballeira E. Analysis of factors that influence the maximum number of repetitions in two upper-body resistance exercises: curl biceps and bench press. J Strength Cond Res. 2010 Jun;24(6):1566–72.

48. Boeckh-Behrens WU, Buskies W. Aspectos especiales del control del entrenamiento. Entrenamiento de la fuerza. Editorial Paidotribo; 2005. p. 66–72.

49. Shimano T, Kraemer WJ, Spiering BA, Volek JS, Hatfield DL, Silvestre R, et al. Relationship between the number of repetitions and selected percentages of one repetition maximum in free weight exercises in trained and untrained men. J Strength Cond Res. 2006 Nov;20(4):819–23.

50. Hoeger WWK, Hopkins DR, Barette SL, Hale DF. Relationship between repetitions and selected percentages of one repetition maximum : A comparison between untrained and trained males and females. J Appl Sport Sci Res. 1990;4(2):47–54.

51. Pick J, Becque MD. The Relationship Between Training Status and Intensity on Muscle Activation and Relative Submaximal Lifting Capacity During the Back Squat. J Strength Cond Res. 2000;14(2):175–81.

52. Willardson JM. The application of training to failure in periodized multiple-set resistance exercise programs. J Strength Cond Res. 2007;21(2):628–31.

53. Baechle TR, Earle RW. Essentials of strength training and conditioning. 3rd ed. Champaing, IL: Human Kinetics; 2008.

54. Fisher J, Steele J, Bruce-Low S, Smith D. Evidence-based resistance training recommendations. Med Sport. 2011 Sep 1;15(3):147–62.

55. Willardson JM, Burkett LN. A comparison of 3 different rest intervals on the exercise volume completed during a workout. J Strength Cond Res. 2005 Feb;19(1):23–6.

56. Willardson JM, Burkett LN. The effect of different rest intervals between sets on volume components and strength gains. J Strength Cond Res. 2008 Jan;22(1):146–52.

57. Sakamoto A, Sinclair PJ. Effect of movement velocity on the relationship between training load and the number of repetitions of bench press. J Strength Cond Res. 2006 Aug;20(3):523–7.

58. Tan B. Manipulating Resistance Training Program Variables to Optimize Maximum Strength in Men: A Review. J Strength Cond Res. 1999;13(3):289–304.

59. Spiering BA, Kraemer WJ, Anderson JM, Armstrong LE, Nindl BC, Volek JS, et al. Resistance exercise biology. Sport Med. 2008;38(7):527–40.

60. Stone MH, Jeff Chandler T, Conley MS, Kramer JB, Stone ME. Training to muscular failure: Is it necessary? Strength Cond J. 1996;18(3):44–8.

61. Steele J, Fisher J, McGuff D, Bruce-Low S, Smith D. Resistance training to momentary muscular failure improves cardiovascular fitness in humans: A review of acute physiological responses and chronic physiological adaptations. JEPonline. 2012;15(3):53–80.

62. Hoffman J. Physiological aspects of sport training and performance. Champaign, IL: Human Kinetics; 2002.

63. Poliquin C. Five steps to increasing the effectiveness of your strength training program. Natl Strength Cond Assoc J. 1988;10(3):34–9.

64. Haff GG, Whitley A, McCoy LB, O'Bryant HS, Kilgore JL, Haff EE, et al. Effects of different set configurations on barbell velocity and displacement during a clean pull. J Strength Cond Res. 2003 Feb;17(1):95–103.

65. Haff GG, Hobbs RT, Haff EE, Sands WA, Pierce KC, Stone MH. Cluster training: A novel method for introducing training program variation. Strength Cond J. 2008 Feb;30(1):67–76.

66. Gorostiaga EM, Navarro-Amézqueta I, Calbet JAL, Hellsten Y, Cusso R, Guerrero M, et al. Energy metabolism during repeated sets of leg press exercise leading to failure or not. PLoS One. 2012;7(7):1–9.

67. Oliver JM, Kreutzer A, Jenke SC, Phillips MD, Mitchell JB, Jones MT. Velocity drives greater power observed during back squat using cluster sets. J Strength Cond Res. 2016 Jan;30(1):235–43.

68. Hardee JP, Travis Triplett N, Utter AC, Zwetsloot KA, Mcbride JM. Effect of interrepetition rest on power output in the power clean. J Strength Cond Res. 2012;26(4):883–9.

69. Oliver JM, Jagim AR, Sanchez AC, Mardock MA, Kelly KA, Meredith HJ, et al. Greater gains in strength and power with intraset rest intervals in hypertrophic training. J Strength Cond Res. 2013 Nov;27(11):3116–31.

70. Izquierdo M, Ibañez J, González-Badillo JJ, Häkkinen K, Ratamess NA, Kraemer WJ, et al. Differential effects of strength training leading to failure versus not to failure on hormonal responses, strength, and muscle power gains. J Appl Physiol. 2006 May;100(5):1647–56.

71. Sánchez-Medina L, González-Badillo JJ. Velocity loss as an indicator of neuromuscular fatigue during resistance training. Med Sci Sports Exerc. 2011 Sep;43(9):1725–34.

72. Baker DG, Newton RU. Change in power output across a high-repetition set of bench throws and jump squats in highly trained athletes. J Strength Cond Res. 2007;21(4):1007–11.

73. Izquierdo M, González-Badillo JJ, Häkkinen K, Ibáñez J, Kraemer WJ, Altadill A, et al. Effect of loading on unintentional lifting velocity declines during single sets of repetitions to failure during upper and lower extremity muscle actions. Int J Sports Med. 2006 Sep;27(9):718–24.

74. Gorostiaga EM, Navarro-Amézqueta I, Calbet JAL, Sánchez-Medina L, Cusso R, Guerrero M, et al. Blood ammonia and lactate as markers of muscle metabolites during leg press exercise. J Strength Cond Res. 2014 Apr 14;

75. Hansen KT, Cronin JB, Newton MJ. The effect of cluster loading on force, velocity, and power during ballistic jump squat training. Int J Sports Physiol Perform. 2011 Dec;6(4):455–68.

76. García-Ramos A, Padial P, Haff GG, Argüelles-Cienfuegos J, García-Ramos M, Conde-Pipó J, et al. Effect of different inter-repetition rest periods on barbell velocity loss during the ballistic bench press exercise. J Strength Cond Res. 2015;1.

77. Iglesias-Soler E, Carballeira E, Sánchez-Otero T, Mayo X, Jiménez A, Chapman ML. Acute effects of distribution of rest between repetitions. Int J Sports Med. 2012 May;33(5):351–8.

78. Lawton TW, Cronin JB, Lindsell RP. Effect of interrepetition rest intervals on weight training repetition power output. J Strength Cond Res. 2006 Feb;20(1):172–6.

79. Moreno SD, Brown LE, Coburn JW, Judelson DA. Effect of cluster sets on plyometric jump power. J Strength Cond Res. 2014 Sep 17;28(9):2424–8.

80. Iglesias-Soler E, Carballeira E, Sánchez-Otero T, Mayo X, Fernández-del-Olmo M. Performance of maximum number of repetitions with cluster-set configuration. Int J Sports Physiol Perform. 2014 Jul 23;9(4):637–42.

81. Keogh JWL, Wilson GJ, Weatherby RP. A Cross-sectional comparison of different resistance training techniques in the bench press. J Strength Cond Res. 1999;13(3):247.

82. Denton J, Cronin JB. Kinematic, kinetic, and blood lactate profiles of continuous and intraset rest loading schemes. J Strength Cond Res. 2006 Aug;20(3):528–34.

83. Goto K, Ishii N, Kizuka T, Takamatsu K. The impact of metabolic stress on hormonal responses and muscular adaptations. Med Sci Sports Exerc. 2005 Jun;37(6):955–63.

84. Oliver JM, Kreutzer A, Jenke S, Phillips MD, Mitchell JB, Jones MT. Acute response to cluster sets in trained and untrained men. Eur J Appl Physiol. 2015 Nov;115(11):2383–93.

85. Häkkinen K, Pakarinen A. Acute hormonal responses to two different fatiguing heavy-resistance protocols in male athletes. J Appl Physiol. 1993;74(2):882–7.

86. Takarada Y, Takazawa H, Sato Y, Takebayashi S, Tanaka Y, Ishii N. Effects of resistance exercise combined with moderate vascular occlusion on muscular function in humans. J Appl Physiol. 2000;88(6):2097–106.

87. Gandevia SC, Herbert RD, Leeper JB. Voluntary activation of human elbow flexor muscles during maximal concentric contractions. J Physiol. 1998;512(2):595–602.

88. Haff G, Burgess S, Stone M. Cluster training : Theoretical and practical applications for the strength and conditioning professional. UK Strength Cond Assoc. 2008;12(July):12–25.

89. Moritani T, Sherman WM, Shibata M, Matsumoto T, Shinohara M. Oxygen availability and motor unit activity in humans. Eur J Appl Physiol Occup Physiol. 1992;64(6):552–6.

90. Rotto DM, Kaufman MP. Effect of metabolic products of muscular contraction on discharge of group III and IV afferents. J Appl Physiol. 1988;64(6):2306–13.

91. Joy JM, Oliver JM, Mccleary SA, Lowery RP, Wilson JM. Power output and electromyography activity of the back squat exercise with cluster sets. J Sport Sci. 2013;1:37–45.

92. Folland JP, Irish CS, Roberts JC, Tarr JE, Jones DA. Fatigue is not a necessary stimulus for strength gains during resistance training. Br J Sports Med. 2002 Oct;36(5):370–3; discussion 374.

93. Iglesias-Soler E, Mayo X, Río-Rodríguez D, Carballeira E, Fariñas J, Fernández-Del-Olmo M. Inter-repetition rest training and traditional set configuration produce similar strength gains without cortical adaptations. J Sports Sci. 2015 Dec 2;0414(December):1–12.

94. Drinkwater EJ, Lawton TW, Lindsell RP, Pyne DB, Hunt PH, McKenna MJ. Training leading to repetition failure enhances bench press strength gains in elite junior athletes. J Strength Cond Res. 2005 May;19(2):382–8.

95. Rooney KJ, Herbert RD, Balnave RJ. Fatigue contributes to the strength training stimulus. Med Sci Sports Exerc. 1994 Sep;26(9):1160–4.

96. Malik M. Time domain measurements of heart rate variability. Card Electrophysiol Rev. 1997;3:329–34.

97. La Rovere MT, Bigger JT, Marcus FI, Mortara A, Schwartz PJ. Baroreflex sensitivity and heart-rate variability in prediction of total cardiac mortality after myocardial infarction. ATRAMI. Lancet. 1998 Feb 14;351(9101):478–84.

98. Malik M. 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. Circulation. 1996 Mar 1;93(5):1043–65.

99. Kuusela TA, Jartti TT, Tahvanainen KUO, Kaila TJ. Nonlinear methods of biosignal analysis in assessing terbutaline-induced heart rate and blood pressure changes. Am J Physiol Heart Circ Physiol. 2002;282:H773–83.

100. Arai Y, Saul JP, Albrecht P, Hartley LH, Lilly LS, Cohen RJ, et al. Modulation of cardiac autonomic activity during and immediately after exercise. Am J Physiol. 1989 Jan;256(1 Pt 2):H132–41.

101. Trevizani GA, Peçanha T, Nasario-Junior O, Vianna JM, Silva LP, Nadal J. Cardiac autonomic responses after resistance exercise in treated hypertensive subjects. Front Physiol. 2015;6(September):1–7.

102. Iglesias-Soler E, Boullosa DA, Carballeira E, Sánchez-Otero T, Mayo X, Castro-Gacio X, et al. Effect of set configuration on hemodynamics and cardiac autonomic modulation after high-intensity squat exercise. Clin Physiol Funct Imaging. 2014 Apr 29;35(4):250–7.

103. Okuno NM, Pedro RE, Leicht AS, de Paula Ramos S, Nakamura FY. Cardiac autonomic recovery after a single session of resistance exercise with and without vascular occlusion. J Strength Cond Res. 2013 Apr 25;28(4):1143–50.

104. de Freitas Brito A, Brasileiro-Santos M do S, Coutinho de Oliveira CV, Sarmento da Nóbrega TK, Lúcia de Moraes Forjaz C, da Cruz Santos A. High-intensity resistance exercise promotes postexercise hypotension greater than moderate intensity and affects cardiac autonomic responses in women who are hypertensive. J Strength Cond Res. 2015 Dec;29(12):3486–93.

105. Lima AHRDA, Forjaz CLDM, Silva GQ de M, Menêses AL, Silva AJMR, Ritti-Dias RM. Acute effect of resistance exercise intensity in cardiac autonomic modulation after exercise. Arq Bras Cardiol. 2011 Jun;96(6):498–503.

106. Figueiredo T, Willardson JM, Miranda H, Bentes CM, Reis VM, Simão R. Influence of Load Intensity on Postexercise Hypotension and Heart Rate Variability after a Strength Training Session. J Strength Cond Res. 2015;29(10):2941–8.

107. 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 May;24(5):1313–20.

108. George DT, Nutt DJ, Walker W V, Porges SW, Adinoff B, Linnoila M. Lactate and hyperventilation substantially attenuate vagal tone in normal volunteers. A possible mechanism of panic provocation? Arch Gen Psychiatry. 1989 Feb;46(2):153–6.

109. Yeragani VK, Srinivasan K, Balon R, Berchou R. Effects of lactate on cross-spectral analysis of heart rate, blood pressure, and lung volume in normal volunteers. Psychiatry Res. 1996 Feb 28;60(1):77–85.

110. Buchheit M, Laursen PB, Ahmaidi S. Parasympathetic reactivation after repeated sprint exercise. Am J Physiol Heart Circ Physiol. 2007;293(1):H133–41.

111. Kaikkonen P, Hynynen E, Mann T, Rusko H, Nummela A. Can HRV be used to evaluate training load in constant load exercises? Eur J Appl Physiol. 2010;108(3):435–42.

112. Seiler S, Haugen O, Kuffel E. Autonomic recovery after exercise in trained athletes: intensity and duration effects. Med Sci Sports Exerc. 2007 Aug;39(8):1366–73.

113. Machado-Vidotti H, Mendes RG, Simões RP, Castello-Simões V, Catai AM, Borghi-Silva A. Cardiac autonomic responses during upper versus lower limb resistance exercise in healthy elderly men. Brazilian J Phys Ther. 2014;18(1):9–18.

114. Fadel PJ, Ogoh S, Keller DM, Raven PB. Recent insights into carotid baroreflex function in humans using the variable pressure neck chamber. Exp Physiol. 2003 Nov;88(6):671–80.

115. Lipman RD, Salisbury JK, Taylor JA. Spontaneous Indices Are Inconsistent With Arterial Baroreflex Gain. Hypertension. 2003;42(4):481–7.

116. Cowley AW, Liard JF, Guyton AC. Role of baroreceptor reflex in daily control of arterial blood pressure and other variables in dogs. Circ Res. 1973;32(5):564–76.

117. Raven PB, Fadel PJ, Ogoh S. Arterial baroreflex resetting during exercise: a current perspective. Exp Physiol. 2006 Jan;91(1):37–49.

118. Kent BB, Drane JW, Blumenstein B, Manning JW. A mathematical model to assess changes in the baroreceptor reflex. Cardiology. 1972 Jan;57(5):295–310.

119. Bristow JD, Honour AJ, Pickering GW, Sleight P, Smyth HS. Diminished baroreflex sensitivity in high blood pressure. Circulation. 1969 Jan 1;39(1):48–54.

120. Reyes del Paso GA, Hernández JA, González MI. Differential analysis in the time domain of the baroreceptor cardiac reflex sensitivity as a function of sequence length. Psychophysiology. 2004 May;41(3):483–8.

121. La Rovere MT, Pinna GD, Raczak G. Baroreflex sensitivity: measurement and clinical implications. Ann Noninvasive Electrocardiol. 2008 Apr;13(2):191–207.

122. Parati G, Di Rienzo M, Mancia G. Dynamic modulation of baroreflex sensitivity in health and disease. Ann N Y Acad Sci. 2001 Jun;940:469–87.

123. Parlow J, Viale JP, Annat G, Hughson R, Quintin L. Spontaneous cardiac baroreflex in humans. Comparison with drug-induced responses. Hypertension. 1995 May;25(5):1058–68.

124. Bertinieri G, Di Rienzo M, Cavallazzi A, Ferrari AU, Pedotti A, Mancia G. Evaluation of baroreceptor reflex by blood pressure monitoring in unanesthetized cats. Am J Physiol. 1988 Feb;254(2 Pt 2):H377–83.

125. Robbe HW, Mulder LJ, Rüddel H, Langewitz WA, Veldman JB, Mulder G. Assessment of baroreceptor reflex sensitivity by means of spectral analysis. Hypertension. 1987 Nov 1;10(5):538–43.

126. Kim SY, Euler DE. Baroreflex sensitivity assessed by complex demodulation of cardiovascular variability. Hypertension. 1997;29(5):1119–25.

127. Pickering TG, Gribbin B, Petersen ES, Cunningham DJ, Sleight P. Effects of autonomic blockade on the baroreflex in man at rest and during exercise. Circ Res. 1972 Feb;30(2):177–85.

128. Ogoh S, Fisher JP, Dawson EA, White MJ, Secher NH, Raven PB. Autonomic nervous system influence on arterial baroreflex control of heart rate during exercise in humans. J Physiol. 2005 Jul 15;566(Pt 2):599–611.

129. Blaber AP, Yamamoto Y, Hughson RL. Methodology of spontaneous baroreflex relationship assessed by surrogate data analysis. Am J Physiol. 1995 Apr;268(4 Pt 2):H1682–7.

130. Kaushal P, Taylor JA. Inter-relations among declines in arterial distensibility, baroreflex function and respiratory sinus arrhythmia. J Am Coll Cardiol. 2002;39(9):1524–30.

131. Matsuda T, Bates JN, Lewis SJ, Abboud FM, Chapleau MW. Modulation of baroreceptor activity by nitric oxide and S-nitrosocysteine. Circ Res. 1995 Mar 1;76(3):426–33.

132. Piepoli M, Sleight P, Leuzzi S, Valle F, Spadacini G, Passino C, et al. Origin of respiratory sinus arrhythmia in conscious humans. An important role for arterial carotid baroreceptors. Circulation. 1997;95(7):1813–21.

133. Kenney MJ, Seals DR. Postexercise hypotension. Key features, mechanisms, and clinical significance. Hypertension. 1993 Nov 1;22(5):653–64.

134. Melo CM, Alencar Filho AC, Tinucci T, Mion D, Forjaz CLM. Postexercise hypotension induced by low-intensity resistance exercise in hypertensive women receiving captopril. Blood Press Monit. 2006 Aug;11(4):183–9.

135. Brown SP, Clemons JM, He Q, Liu S. Effects of resistance exercise and cycling on recovery blood pressure. J Sports Sci. 1994 Oct;12(5):463–8.

136. 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 Jan;79(2):148–54.

137. Morais PK, Campbell CSG, Sales MM, Motta DF, Moreira SR, Cunha VNC, et al. Acute resistance exercise is more effective than aerobic exercise for 24h blood pressure control in type 2 diabetics. Diabetes Metab. 2011 Apr;37(2):112–7.

138. Mota MR, Pardono E, Lima LCJ, Arsa G, Bottaro M, Campbell CSG, et al. Effects of treadmill running and resistance exercises on lowering blood pressure during the daily work of hypertensive subjects. J Strength Cond Res. 2009 Nov;23(8):2331–8.

139. Chen C, Bonham AC. Postexercise hypotension: central mechanisms. Exerc Sport Sci Rev. 2010 Jul;38(3):122–7.

140. Thompson PD, Crouse SF, Goodpaster B, Kelley D, Moyna N, Pescatello L. The acute versus the chronic response to exercise. Med Sci Sports Exerc. 2001 Jun;33(6 Suppl):S438–45; discussion S452–3.

141. Tibana RA, de Sousa NMF, da Cunha Nascimento D, Pereira GB, Thomas SG, Balsamo S, et al. Correlation between acute and chronic 24-Hour blood pressure response to resistance training in adult women. Int J Sports Med. 2015 Aug 21;36(1):82–9.

142. Mediano FMF, Paravidino V, Simão R, Pontes FL, Polito MD. Subacute behavior of the blood pressure after power training in controlled hypertensive individuals. Rev Bras Med do Esporte. 2005;11(6):307–9.

143. Scher LML, Ferriolli E, Moriguti JC, Scher R, Lima NKC. The effect of different volumes of acute resistance exercise on elderly individuals with treated hypertension. J Strength Cond Res. 2011 Apr;25(4):1016–23.

144. Cucato GG, Ritti-Dias RM, Wolosker N, Santarem JM, Jacob Filho W, Forjaz CLDM. Post-resistance exercise hypotension in patients with intermittent claudication. Clinics. 2011;66(2):221–6.

145. Hardy DO, Tucker LA. The effects of a single bout of strength training on ambulatory blood pressure levels in 24 mildly hypertensive men. Am J Health Promot. 1998;13(2):69–72.

146. Brito AF, Alves NFB, Araújo AS, Gonçalves MCR, Silva AS. Active intervals between sets of resistance exercises potentiate the magnitude of postexercise hypotension in elderly hypertensive women. J Strength Cond Res. 2011 Nov;25(11):3129–36.

147. Halliwill JR, Buck TM, Lacewell AN, Romero SA. Postexercise hypotension and sustained postexercise vasodilatation: what happens after we exercise? Exp Physiol. 2013 Jan;98(1):7–18.

148. Taylor CE, Jones H, Zaregarizi M, Cable NT, George KP, Atkinson G. Blood pressure status and post-exercise hypotension: an example of a spurious correlation in hypertension research? J Hum Hypertens. Nature Publishing Group; 2010;24(9):585–92.

149. Halliwill JR, Taylor JA, Eckberg DL. Impaired sympathetic vascular regulation in humans after acute dynamic exercise. J Physiol. 1996 Aug 15;495 (Pt 1:279–88.

150. Floras JS, Sinkey CA, Aylward PE, Seals DR, Thoren PN, Mark AL. Postexercise hypotension and sympathoinhibition in borderline hypertensive men. Hypertension. 1989 Jul;14(1):28–35.

151. Floras JS, Senn BL. Absence of post exercise hypotension and sympathoinhibition in normal subjects: additional evidence for increased sympathetic outflow in borderline hypertension. Can J Cardiol. 1991;7(6):253–8.

152. Barrett-O'Keefe Z, Kaplon RE, Halliwill JR. Sustained postexercise vasodilatation and histamine receptor activation following small muscle-mass exercise in humans. Exp Physiol. 2013 Jan;98(1):268–77.

153. McCord JL, Halliwill JR. H1 and H2 receptors mediate postexercise hyperemia in sedentary and endurance exercise-trained men and women. J Appl Physiol. 2006 Dec;101(6):1693–701.

154. Bennett T, Wilcox RG, Macdonald IA. Post-exercise reduction of blood pressure in hypertensive men is not due to acute impairment of baroreflex function. Clin Sci. 1984;67(1):97–103.

155. Hara K, Floras JS. Effects of naloxone on hemodynamics and sympathetic activity after exercise. J Appl Physiol. 1992;73(5):2028–35.

156. Lockwood JM, Wilkins BW, Halliwill JR. H1 receptor-mediated vasodilatation contributes to postexercise hypotension. J Physiol. 2005 Mar 1;563(Pt 2):633–42.

157. McCord JL, Beasley JM, Halliwill JR. H2-receptor-mediated vasodilation contributes to postexercise hypotension. J Appl Physiol. 2006 Jan;100(1):67–75.

158. Lockwood JM, Pricher MP, Wilkins BW, Holowatz LA, Halliwill JR. Postexercise hypotension is not explained by a prostaglandin-dependent peripheral vasodilation. J Appl Physiol. 2005 Mar;98(2):447–53.

159. Halliwill JR, Minson CT, Joyner MJ. Effect of systemic nitric oxide synthase inhibition on postexercise hypotension in humans. J Appl Physiol. 2000;89(5):1830–6.

160. Halliwill JR. Mechanisms and clinical implications of post-exercise hypotension in humans. Exerc Sport Sci Rev. 2001 Apr;29(2):65–70.

161. Brito LC, Queiroz ACC, Forjaz CLM. Influence of population and exercise protocol characteristics on hemodynamic determinants of post-aerobic exercise hypotension. Brazilian J Med Biol Res. 2014;47(8):626–36.

162. Raine NM, Cable NT, George KP, Campbell IG. The influence of recovery posture on post-exercise hypotension in normotensive men. Med Sci Sports Exerc. 2001 Mar;33(3):404–12.

163. Takahashi T, Hayano J, Okada A, Saitoh T, Kamiya A. Effects of the muscle pump and body posture on cardiovascular responses during recovery from cycle exercise. Eur J Appl Physiol. 2005;94:576–83.

164. Hagberg JM, Montain SJ, Martin WH. Blood pressure and hemodynamic responses after exercise in older hypertensives. J Appl Physiol. 1987;63(1):270–6.

165. Rueckert PA, Slane PR, Lillis DL, Hanson P. Hemodynamic patterns and duration of post-dynamic exercise hypotension in hypertensive humans. Med Sci Sports Exerc. 1996 Jan;28(1):24–32.

166. Franklin PJ, Green DJ, Cable NT. The influence of thermoregulatory mechanisms on post-exercise hypotension in humans. J Physiol. 1993 Oct;470:231–41.

167. Charkoudian N, Halliwill JR, Morgan BJ, Eisenach JH, Joyner MJ. Influences of hydration on post-exercise cardiovascular control in humans. J Physiol. 2003 Oct 15;552(Pt 2):635–44.

168. Lynn BM, Minson CT, Halliwill JR. Fluid replacement and heat stress during exercise alter post-exercise cardiac haemodynamics in endurance exercise-trained men. J Physiol. 2009;587:3605–17.

169. Bermudes AMLDM, 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 Jan;82(1):65–71, 57–64.

170. Prista A, Macucule CF, Queiroz ACC, Silva ND, Cardoso CG, Tinucci T, et al. A bout of resistance exercise following the 2007 AHA guidelines decreases asleep blood pressure in Mozambican men. J Strength Cond Res. 2013 Mar;27(3):786–92.

171. Anunciação PG, Casonatto J, Polito MD. Blood pressure responses and heart rate variability after resistance exercise with different intensities and same workload. Int Sport J. 2011;12(2):53–67.

172. Simão 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 Nov;19(4):853–8.

173. de Salles BF, Maior AS, Polito M, Novaes J, Alexander J, Rhea M, et al. Influence of rest interval lengths on hypotensive response after strength training sessions performed by older men. J Strength Cond Res. 2010 Nov;24(11):3049–54.

174. Jannig PR, Cardoso AC, Fleischmann E, Coelho CW, Carvalho T. Influência da ordem de execução de exercícios resistidos na hipotensão pós-exercício em idosos hipertensos. Rev Bras Med do Esporte. 2009 Oct;15(5):338–41.

175. Moraes MR, Bacurau RFP, Ramalho JDS, Reis FCG, Casarini DE, Chagas JR, et al. Increase in kinins on post-exercise hypotension in normotensive and hypertensive volunteers. Biol Chem. 2007 May;388(5):533–40.

176. Lizardo J, Simões H. Efeitos de diferentes sessões de exercícios resistidos sobre a hipotensõo pós-exercício. Rev Bras Fisioter. 2005;9(3):289–95.

177. Hill DW, Collins MA, Cureton KJ, DeMello JJ. Blood pressure response after weight training exercise. J Appl Sport Sci Res. 1989;3(2):44–7.

178. 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 Apr;25(4):516–21.

179. Duncan MJ, Birch SL, Oxford SW. The effect of exercise intensity on postresistance exercise hypotension in trained men. J Strength Cond Res. 2014 Jun 22;28(6):1706–13.

180. Canuto PMBC, Nogueira IDB, Cunha ES, Ferreira GMF, Mendonça KMPP, Costa FA, et al. Influência do treinamento resistido realizado em intensidades diferentes e mesmo volume de trabalho sobre a pressão arterial de idosas hipertensas. Rev Bras Med do Esporte. 2011 Aug;17(4):246–9.

181. Gurjão ALD, Salvador EP, Cyrino ES, Gerage AM, Schiavoni D, Gobbi S. Respostas pressóricas pós-exercícios com pesos executados em diferentes sobrecargas por mulheres normotensas. Rev Bras Med do Esporte. 2009 Feb;15(1):14–8.

182. Focht BC, Koltyn KF. Influence of resistance exercise of different intensities on state anxiety and blood pressure. Med Sci Sports Exerc. 1999 Mar;31(3):456–63.

183. Roltsch MH, Mendez T, Wilund KR, Hagberg JM. Acute resistive exercise does not affect ambulatory blood pressure in young men and women. Med Sci Sports Exerc. 2001 Jun;33(6):881–6.

184. Fisher MM. The effect of resistance exercise on recovery blood pressure in normotensive and borderline hypertensive women. J Strength Cond Res. 2001 May;15(2):210–6.

185. Veloso J, Polito MD, Riera T, Celes R, Vidal JC, Bottaro M. Effects of rest interval between exercise sets on blood pressure after resistance exercises. Arq Bras Cardiol. 2010;94(4):512–8.

186. Romero F, Caperuto E, Costa Rosa L. Efeitos de diferentes métodos de exercícios resistidos sobre o comportamento hemodinâmico. Rev Bras Ciência e Mov. 2005;13(2):7–15.

187. Polito MD, Rosa CC, Schardong P. Acute cardiovascular responses on knee extension at different performance modes. Rev Bras Med do Esporte. 2004;10(3):177–80.

188. Menêses AL, Forjaz CL de M, Silva GQ de M, Lima AHR de A, Farah BQ, Lins Filho O de L, et al. Post exercise cardiovascular effects of different resistance exercise protocols for trunk and upper limbs. Mot Rev Educ Física. 2011 Dec;17(4):667–74.

189. Bentes CM, Costa PB, Neto GR, Costa e Silva G V., de Salles BF, Miranda HL, et al. Hypotensive effects and performance responses between different resistance training intensities and exercise orders in apparently health women. Clin Physiol Funct Imaging. 2015 May 1;35(3):185–90.

190. Paz G, Maia M, Bentes CM, Figueiredo T, Salerno V, Simão R, et al. Effect of agonist-antagonist paired set training vs. traditional set training on post-resistance exercise hypotension. JEPonline. 2014;17(6):13–23.

191. Polito MD, da Nóbrega ACL, Farinatti P. Blood pressure and forearm blood flow after multiple sets of a resistive exercise for the lower limbs. Blood Press Monit. 2011 Aug;16(4):180–5.

192. Moraes Miguel F, Alexandre Grings L, Borges Pereira G, Diego Leite R, Vieira A, Frade de Sousa NM, et al. Different cardiovascular responses to a resistance training session in hypertensive women receiving propanolol compared with normotensive controls. ScientificWorldJournal. 2012 Jan;2012:913271.

193. Rodriguez D, Polito MD, Bacurau RFP, Prestes J, Pontes Jr, FL. Effect of different resistance exercise methods on post-exercise blood pressure. Int J Exerc Sci. 2008;1(4):153–62.

194. Faraji H, Bab L, Ardeshiri H. Effects of resistance exercise intensity and volume on postexercise hypotensive respones. Brazilian J Biomotricity. 2010;4(1):65–73.

195. Simões GC, Moreira SR, Kushnick MR, Simões HG, Campbell CSG. Postresistance exercise blood pressure reduction is influenced by exercise intensity in type-2 diabetic and nondiabetic individuals. J Strength Cond Res. 2010 May;24(5):1277–84.

196. Polito MD, Simão R, Senna GW, Farinatti P de TV. Hypotensive effects of resistance exercises performed at different intensities and same work volumes. Rev Bras Med. 2003;9(2):74–7.

197. Koltyn KF, Raglin JS, O’Connor PJ, Morgan WP. Influence of weight training on state anxiety, body awareness and blood pressure. Int J Sports Med. 1995 May;16(4):266–9.

198. Bangsbo J, Hellsten Y. Muscle blood flow and oxygen uptake in recovery from exercise. Acta Physiol Scand. 1998 Mar;162(3):305–12.

199. Queiroz ACC, Rezk CC, Teixeira L, Tinucci T, Mion D, Forjaz CLM. Gender influence on post-resistance exercise hypotension and hemodynamics. Int J Sports Med. 2013 Nov;34(11):939–44.

200. Collins MA, Hill DW, Cureton KJ, DeMello JJ. Plasma volume change during heavy-resistance weight lifting. Eur J Appl Physiol Occup Physiol. 1986;55:44–8.

201. Bush JA, Kraemer WJ, Mastro AM, Triplett-McBride NT, Volek JS, Putukian M, et al. Exercise and recovery responses of adrenal medullary neurohormones to heavy resistance exercise. Medicine and science in sports and exercise. 1999.

202. Cléroux J, Kouamé N, Nadeau A, Coulombe D, Lacourcière Y. Aftereffects of exercise on regional and systemic hemodynamics in hypertension.. Hypertension. 1992 Feb [cited 2012 Jul 30].

203. Anrep G V, Barsoum GS, Salama S, Souidan Z. Liberation of histamine during reactive hyperaemia and muscle contraction in man. J Physiol. 1944;103(3):297–305.

204. Endo Y, Nakamura M, Nitta Y, Kumagai K. Effects of macrophage depletion on the induction of histidine decarboxylase by lipopolysaccharide, interleukin 1 and tumour necrosis factor. Br J Pharmacol. 1995;114:187–93.

205. Endo Y, Tabata T, Kuroda H, Tadano T, Matsushima K, Watanabe M. Induction of histidine decarboxylase in skeletal muscle in mice by electrical stimulation, prolonged walking and interleukin-1. J Physiol. 1998;509:587–98.

206. Fahs CA, Heffernan KS, Fernhall B. Hemodynamic and vascular response to resistance exercise with l-arginine. Med Sci Sports Exerc. 2009;41(4):773–9.

207. Emhoff C-AW, Barrett-O’Keefe Z, Padgett RC, Hawn JA, Halliwill JR. Histamine-receptor blockade reduces blood flow but not muscle glucose uptake during postexercise recovery in humans. Exp Physiol. 2011;96:664–73.

208. Marcora SM, Staiano W, Manning V. Mental fatigue impairs physical performance in humans. J Appl Physiol. 2009;106:857–64.

209. Williamson JW, McColl R, Mathews D, Mitchell JH, Raven PB, Morgan WP. Brain activation by central command during actual and imagined handgrip under hypnosis. J Appl Physiol. 2002 Mar;92(3):1317–24.

210. Winchester PK, Williamson JW, Mitchell JH. Cardiovascular responses to static exercise in patients with Brown-Sequard syndrome. J Physiol. 2000;527 Pt 1:193–202.

211. Cafarelli E, Layton-Wood J. Effect of vibration on force sensation in fatigued muscle. Med Sci Sports Exerc. 1986 Oct;18(5):516–21.

212. St Clair Gibson A, Lambert E V, Rauch LHG, Tucker R, Baden DA, Foster C, et al. The role of information processing between the brain and peripheral physiological systems in pacing and perception of effort. Sport Med. 2006;36(8):705–22.

213. Robertson RJ, Goss FL, Rutkowski J, Lenz B, Dixon C, Timmer J, et al. Concurrent validation of the OMNI perceived exertion scale for resistance exercise. Med Sci Sports Exerc. 2003 Feb;35(2):333–41.

214. Naclerio F, Chapman M, Larumbe-Zabala E. Use of rate of perceived exertion scales in resistance training: A comment on Mayo, Iglesias-Soler, and Fernández-del-Olmo (2014). Percept Mot Skills. 2015 Oct;121(2):490–3.

215. Tibana RA, Vieira DCL, Tajra V, Bottaro M, de Salles BF, Willardson JM, et al. Effects of rest interval length on Smith machine bench press performance and perceived exertion in trained men. Percept Mot Skills. 2013 Dec;117(3):682–95.

216. Gearhart RE, Goss FL, Lagally KM, Jakicic JM, Gallagher J, Robertson RJ. Standardized scaling procedures for rating perceived exertion during resistance exercise. J Strength Cond Res. 2001 Aug;15(3):320–5.

217. Noble BJ, Robertson RJ. Perceived exertion. Champaign, IL: Human Kinetics; 1996.

218. Lagally KM, Robertson RJ. Construct validity of the OMNI resistance exercise scale. J Strength Cond Res. 2006 May;20(2):252–6.

219. Hollander DB, Durand RJ, Trynicki JL, Larock D, Castracane VD, Hebert EP, et al. RPE, pain, and physiological adjustment to concentric and eccentric contractions. Med Sci Sports Exerc. 2003 Jun;35(6):1017–25.

220. Duncan MJ, Al-Nakeeb Y, Scurr J. Perceived exertion is related to muscle activity during leg extension exercise. Res Sports Med. 2006;14(3):179–89.

221. Buckley JP, Borg GA V. Borg's scales in strength training; from theory to practice in young and older adults. Appl Physiol Nutr Metab. 2011 Oct;36(5):682–92.

222. Hackett DA, Johnson NA, Halaki M, Chow C. A novel scale to assess resistance-exercise effort. J Sports Sci. 2012 Jan;30(13):1405–13.

223. Pincivero DM, Gear WS. Quadriceps activation and perceived exertion during a high intensity, steady state contraction to failure. Muscle Nerve. 2000 Apr;23(4):514–20.

224. Row BS, Knutzen KM, Skogsberg NJ. Regulating explosive resistance training intensity using the rating of perceived exertion. J Strength Cond Res. 2012 Mar;26(3):664–71.

225. Lagally KM, Amorose AJ, Rock B. Selection of resistance exercise intensity using ratings of perceived exertion from the OMNI-RES. Percept Mot Skills. 2009 Apr;108(2):573–86.

226. Naclerio F, Rodríguez-Romo G, Barriopedro-Moro MI, Jiménez A, Alvar BA, Triplett NT. Control of resistance training intensity by the OMNI perceived exertion scale. J Strength Cond Res. 2011 Jul;25(7):1879–88.

227. Eston R, James H, Evans L. The validity of submaximal ratings of perceived exertion to predict one repetition maximum. J Sport Sci Med. 2009;8:567–73.

228. Pierce K, Rozenek R, Stone MH. Effects of high volume weight training on lactate, heart rate, and perceived exertion. J Strength Cond Res. 1993 Nov;7(4):211–5.

229. Gearhart RF, Lagally KM, Riechman SE, Andrews RD, Robertson RJ. Strength tracking using the OMNI resistance exercise scale in older men and women. J Strength Cond Res. 2009 May;23(3):1011–5.

230. Woods S, Bridge T, Nelson D, Risse K, Pincivero DM. The effects of rest interval length on ratings of perceived exertion during dynamic knee extension exercise. J Strength Cond Res. 2004 Aug;18(3):540–5.

231. Sousa P, Pinheiro F, Monge D, Pires F. Pressão arterial e PSE em múltiplas séries de exercício resistido de diferentes intensidades. Brazilian J Sci Mov. 2010;18(4):26–33.

232. Hardee JP, Lawrence MM, Utter AC, Triplett NT, Zwetsloot KA, McBride JM. Effect of inter-repetition rest on ratings of perceived exertion during multiple sets of the power clean. Eur J Appl Physiol. 2012 Aug;112(8):3141–7.

233. Kraft JA, Green JM, Thompson KR. Session ratings of perceived exertion responses during resistance training bouts equated for total work but differing in work rate. J Strength Cond Res. 2014 Feb;28(2):540–5.

234. Sweet TW, Foster C, McGuigan MR, Brice G. Quantitation of resistance training using the session rating of perceived exertion method. J Strength Cond Res. 2004 Nov;18(4):796–802.

235. Faigenbaum AD, Milliken LA, Cloutier G, Westcott WL. Perceived exertion during resistance exercise by children. Percept Mot Skills. 2004 Apr;98(2):627–37.

236. Vasquez LM, McBride JM, Paul JA, Alley JR, Carson LT, Goodman CL. Effect of resistance exercise performed to volitional failure on ratings of perceived exertion. Percept Mot Skills. 2013 Dec;117(3):881–91.

237. Hiscock DJ, Dawson B, Peeling P. Perceived exertion responses to changing resistance training programming variables. J Strength Cond Res. 2014 Nov 26;

238. Robertson RJ. Perceived exertion for practitioners. Champaign, IL: Human Kinetics; 2004.

239. Polito MD, Simão R, Viveiros LE. Tempo de tensão, percentual de carga e esforço percebido em testes de força envolvendo diferentes repetições máximas. Rev Bras Fisiol do Exerc. 2003;2(3):97–103.

240. Senna G, Willardson JM, de Salles BF, Scudese E, Carneiro F, Palma A, et al. The effect of rest interval length on multi and single-joint exercise performance and perceived exertion. J Strength Cond Res. 2011 Nov;25(11):3157–62.

241. Duncan MJ, Weldon A, Price MJ. The effect of sodium bicarbonate ingestion on back squat and bench press exercise to failure. J Strength Cond Res. 2014 May 11;28(5):1358–66.

242. Pritchett RC, Green JM, Wickwire PJ, Pritchett KL, Kovacs MS. Acute and session RPE responses during resistance training: Bouts to failure at 60% and 90% of 1RM. Soudafrican J Sport Med. 2009;21(1):23–6.

243. Lodo L, Moreira A, Zavanela PM, Newton MJ, McGuigan MR, Aoki MS. Is there a relationship between the total volume of load lifted in bench press exercise and the rating of perceived exertion? J Sports Med Phys Fitness. 2012 Oct;52(5):483–8.

244. Charro MA, Aoki MS, Coutts AJ, Araújo RC, Bacurau RF. Hormonal, metabolic and perceptual responses to different resistance training systems. J Sports Med Phys Fitness. 2010 Jun;50(2):229–34.

245. Lagally KM, Robertson RJ, Gallagher KI, Goss FL, Jakicic JM, Lephart SM, et al. Perceived exertion, electromyography, and blood lactate during acute bouts of resistance exercise. Med Sci Sports Exerc. 2002 Mar;34(3):552–9; discussion 560.

246. Lagally KM, McCaw ST, Young GT, Medema HC, Thomas DQ. Ratings of perceived exertion and muscle activity during the bench press exercise in recreational and novice lifters. J Strength Cond Res. 2004 May;18(2):359–64.

247. Hiscock DJ, Dawson B, Donnelly CJ, Peeling P. Muscle activation, blood lactate, and perceived exertion responses to changing resistance training programming variables. Eur J Sport Sci. 2015 Aug 12;(AUGUST):1–9.

248. Suminski RR, Robertson RJ, Arslanian S, Kang J, Utter AC, DaSilva SG, et al. Perception of Effort During Resistance Exercise. J Strength Cond Res. 1997;11(4):261–5.

249. Kraemer RR, Acevedo EO, Dzewaltowski D, Kilgore JL, Kraemer GR, Castracane VD. Effects of low-volume resistive exercise on beta-endorphin and cortisol concentrations. Int J Sports Med. 1996 Jan;17(1):12–6.

250. Kraemer WJ, Noble BJ, Clark MJ, Culver BW. Physiologic responses to heavy-resistance exercise with very short rest periods. Int J Sports Med. 1987 Aug;8(4):247–52.

251. Thornton MK, Potteiger JA. Effects of resistance exercise bouts of different intensities but equal work on EPOC. Med Sci Sports Exerc. 2002 Apr;34(4):715–22.

252. Costa E, Moreira A, Cavalcanti B, Krinski K, Aoki M. Effect of unilateral and bilateral resistance exercise on maximal voluntary strength, total volume of load lifted, and perceptual and metabolic responses. Biol Sport. 2014;32(1):35–40.

253. Arent SM, Landers DM, Matt KS, Etnier JL. Dose-response and mechanistic issues in the resistance training and affect relationship. J Sport Exerc Psychol. 2005;27(1):92–110.

254. McGuigan MR, Egan AD, Foster C. Salivary cortisol responses and perceived exertion during high intensity and low intensity bouts of resistance exercise. J Sport Sci Med. 2004;3:8–15.

255. Diniz RCR, Martins-Costa HC, Machado SC, Lima F V., Chagas MH. Repetition duration influences ratings of perceived exertion. Percept Mot Skills. 2014 Feb;118(1):261–73.

256. Piras A, Persiani M, Damiani N, Perazzolo M, Raffi M. Peripheral heart action (PHA) training as a valid substitute to high intensity interval training to improve resting cardiovascular changes and autonomic adaptation. Eur J Appl Physiol. 2014;115(4):763–73.

257. Selig SE, Carey MF, Menzies DG, 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 Feb;10(1):21–30.

258. Kraemer WJ, Fry A. Strength testing: development and evaluation of methodology. In: Maud PJ, Foster C, eds. Physiological Assessment of Human Fitness. Human Kinetics, Champaign; 1995. 115–138 p.

259. Pagani M, Lombardi F, Guzzetti S, Rimoldi O, Furlan R, Pizzinelli P, et al. Power spectral analysis of heart rate and arterial pressure variabilities as a marker of sympatho-vagal interaction in man and conscious dog. Circ Res. 1986 Aug;59(2):178–93.

260. Richman JS, Moorman JR. Physiological time-series analysis using approximate entropy and sample entropy. Am J Physiol Heart Circ Physiol. 2000 Jun;278(6):H2039–49.

261. Dias I, Simão R, Novaes J. The resistive exercises influence in different muscular groupments on blood pressure. Fit Perform J. 2007 Mar 1;6(2):71–5.

262. Mayo X, Iglesias-Soler E, Fernández-Del-Olmo M. Effects of set configuration of resistance exercise on perceived exertion. Percept Mot Skills. 2014 Dec 2;119(3):825–37.

263. Piepoli M, Coats AJ, Adamopoulos S, Bernardi L, Feng YH, Conway J, et al. Persistent peripheral vasodilation and sympathetic activity in hypotension after maximal exercise. J Appl Physiol. 1993 Apr 15;75(4):1807–14.

264. Cohen MA, Taylor JA. Short-term cardiovascular oscillations in man: measuring and modelling the physiologies. J Physiol. 2002 Aug 1;542(Pt 3):669–83.

265. Hayano J, Sakakibara Y, Yamada M, Ohte N, Fujinami T, Yokoyama K, et al. Decreased magnitude of heart rate spectral components in coronary artery disease. Its relation to angiographic severity. Circulation. 1990;81:1217–24.

266. Sacha J, Barabach S, Statkiewicz-Barabach G, Sacha K, Müller A, Piskorski J, et al. How to strengthen or weaken the HRV dependence on heart rate - Description of the method and its perspectives. Int J Cardiol. 2013;168(2):1660–3.

267. Sanchez-Medina L, Perez CE, Gonzalez-Badillo JJ. Importance of the propulsive phase in strength assessment. Int J Sports Med. 2010 Feb;31(2):123–9.

268. Yeragani VK, Srinivasan K, Pohl R, Berger R, Balon R, Berchou R. Sodium lactate increases sympathovagal ratios in normal control subjects: spectral analysis of heart rate, blood pressure, and respiration. Psychiatry Res. 1994 Oct;54(1):97–114.

269. Gotshall RW, Aten LA, Yumikura S. Difference in the cardiovascular response to prolonged sitting in men and women. Can J Appl Physiol. 1994;19:215–25.

270. Willardson JM, Burkett LN. The effect of rest interval length on bench press performance with heavy vs. light loads. J Strength Cond Res. 2006 May;20(2):396–9.

271. Day ML, McGuigan MR, Brice G, Foster C. Monitoring exercise intensity during resistance training using the session RPE scale. J Strength Cond Res. 2004 May;18(2):353–8.

272. Simão R, de Salles BF, Figueiredo T, Dias I, Willardson JM. Exercise order in resistance training. Sport Med. 2012 Mar 1;42(3):251–65.

273. Kannankeril PJ, Goldberger JJ. Parasympathetic effects on cardiac electrophysiology during exercise and recovery. Am J Physiol Heart Circ Physiol. 2002;282(6):H2091–8.

274. Rardon DP, Bailey JC. Parasympathetic effects on electrophysiologic properties of cardiac ventricular tissue. J Am Coll Cardiol. 1983;2(6):1200–9.

Appendix A

Abstract of at least 3000 words in an official language

Resumo

O exercicio de forza prevén e mellora varias enfermidades musculoesqueléticas e metabólicas e é recomendado polo *American College of Sports Medicine* e pola *American Heart Association* como unha maneira de revertir patoloxías relacionadas con esas afeccións e para mellorar a calidade de vida. Neste sentido, o exercicio de forza promove certos beneficios para a saúde, como un aumento da masa muscular, da densidade mineral ósea e un aumento do metabolismo da glicose, dos lípidos e das lipoproteínas. Sen embargo, as respostas cardiovasculares agudas e crónicas ao exercicios de forza non son ben coñecidas, a pesares dos esforzos que se teñen realizado últimamente para deseñar adestramentos de forza co fin de mellorar as afeccións de individuos con patoloxías cardiovasculares.

Hoxe en día, sábese que unha sesión de adestramento de forza produce unha redución do control cardíaco parasimpático que é mediado por unha redución da actividade autónoma vagal e por unha redución vagal e glosofarínxea baroreflexa. Esta redución pode significar un efecto perxudicial transitorio en individuos con risco cardiovascular xa que 30 minutos despois dun exercicio existe un risco maior de sufrir un avento cardíaco debido a unha redución da actividade vagal.

Por outro lado, sábese que o adestramento de forza produce de maneira aguda e crónica unha redución da presión arterial, que pode significar unha redución do risco de sufrir un evento cardíaco. Sen embargo, cando o efecto beneficioso do adestramento de forza nesta variable é ben coñecido, o escaso número de estudos realizados provocan un enlentecemento da posible aplicabilidade do adestramento como unha terapia non farmacolóxica para previr ou tratar a hipertensión arterial.

Neste sentido, non hai consenso de como se ten que prescribir o adestramento de forza, principalmente debido a novidade do campo de estudo e a falta de coñecemento aplicable. Existen varios factores que poden contribuír ós efectos do exercicio de forza no sistema cardiovascular, como as características da carga (é dicir, a carga, o volume, etc.), o tipo de exercicio (é dicir, membros superiores en comparación con membros inferiores), ou a forma de prescribilo (é dicir, polo porcentaxe do 1RM, polo número de repeticións ata o fallo, etc.).

Así, as características da carga, o tipo de exercicio usado e a forma de prescribilo determinan e modulan as respostas cardiovasculares de maneira aguda e crónica. Esencialmente, a primeira e a última están interrelacionadas xa que a forma de prescribir o exercicio determina factores como a carga utilizada, as repeticións que se poden realizar ou o descanso empregado, modificando polo tanto o volume total realizable e a relación entre o traballo e a pausa. En resumo, non se coñecen de maneira precisa as respostas de cada variable e como a súa realización contribúe a efectos desexables ou non desexables para a homeotase postexercicio, como a redución do control cardíaco autónomo e reflexo ou a aparición do efecto hipotensivo.

Ademáis, máis aló dos efectos cardiovasculares, o exercicio de forza produce unha resposta perceptiva coñecida como esforzo percibido. O esforzo percibido debe terse en conta xa que controlar a percepción da carga dunha sesión podería permitir regulala e coñecer a implicación fisiolóxica da mesma. Neste sentido, coñecer como os diferentes parámetros da carga e as características de sesión afectan ao esforzo percibido podería axudar a entender no futuro como este está determinado por diferentes procesos mecánicos, metabólicos ou neurais.

En relación a maneira de prescribir o exercicios de forza, varios compoñentes da sesión manipúlanse para maximizar o efecto do adestramento. Tradicionalmente, o compoñente máis importante é o comunmente chamado intensidade, que fai referencia a carga empregada. A súa importancia reside en que determina o número total de repeticións que se poden realizar antes de chegar ao fallo muscular.

A intensidade da carga prescríbese tradicionalmente de dúas maneiras: A máxima manifestación de forza (é dicir, 1RM) e o número máximo de repeticións que e poden facer cun peso submáximo antes do fallo muscular (é dicir, RM). Ambos mecanismos teñen ventaxas e desventaxas que deben terse en conta para maximizar o efecto do programa de forza e para controlar os efectos cardiovasculares e perceptivos que son provocados pola maneira de prescribir a sesión.

Prescribir o exercicio de forza como un porcentaxe do 1RM é recoñecido como o estímulo máis importante para producir cambios nos niveis de forza. Sen embargo, non toda a evidencia apoia a hipótese que adestrar a un porcentaxe do 1RM é importante para as ganancias de forza. Mentres que permite prescribir exercicio a moitos individuos ao mesmo tempo é que pode transformarse fácilmente en valores absolutos, non proporciona ninguna información precisa en relación ao número de repeticións que se poden facer é e dependente do exercicio e ten unha gran variabilidade inter-suxeito, a diferenza de prescribir a través das repeticións máximas (é dicir, RM).

Prescribir a través do número máximo de repeticións, pola contra, só acontece realmente durante a primeira serie, xa que nas posteriores series o individuo é incapaz de manter as repeticións desexadas. Ademáis, adestrar ata o fallo non é necesariamente o mellor estímulo para mellorar os niveis de forza e pode levar ao sobreadestramento ou a lesión.

A prescripción a través da configuración da serie é un xeito alternativo que pode influír nas respostas agudas e crónicas ao exercicio de forza. A configuración da serie fai referencia ao número de repeticións que se fan nunha serie en relación ao número máximo de repeticións que se poderían realizar. Neste sentido, para varios autores é un parámetro de carga independente, recomendando a prescripción de exercicio a través dela.

A configuración da serie pode manipularse de dúas maneiras, unha vez que a intensidade da carga e o volume total téñense escollido: Polo número de repeticións realizadas en cada serie e polo tempo de descanso entre cada serie ou grupo de series. Mentres que o número de repeticións realizadas indica o preto ou lonxe que se está do fallo muscular, e polo tanto da *intensidade do esforzo*, o tempo de descanso entre series indica a reposición metabólica que se quere permitir para unha recuperación máis ou menos completa. As configuracións da serie curtas permiten un rendemento mecánico maior cunha menor participación metabólica, hormonal e neural en comparación coas series longas. Sen embargo, os efectos crónicos das configuracións curtas e longas son comparables nas melloras en forza máxima, resistencia á forza e hipertrofia, con maiores incrementos en potencia con series curtas, especialmente preto da carga de potencia óptima (é dicir, potencia máxima) e co membro superior. Mentres que a maioría dos estudos revelan que non hai diferenzas crónicas no adestramento de forza con configuracións da serie curtas ou longas, as respostas cardiovascular e perceptivas son posiblemente diferentes.

As reducións observadas no control autónomo e barroreflexo do corazón despois do exercicio de forza, débense controlar en determinadas poboacións xa que estas perdas están relacionadas cun aumento transitorio do risco de sufrir un evento cardíaco. A configuración da serie posiblemente permita modular a perda do control cardíaco xa que a implicación glicolítica está relacionada coa retirada vagal, e a duración da serie determina a produción glicolítica da sesión. Ademáis, o estudo da interacción co tipo de exercicio permitirá un maior entendemento da configuración da serie en relación ao control cardíaco, xa que o tipo de exercicio tamén determina a implicación glicolítica.

En relación ao efecto hipotensivo despois do exercicio de forza, parece que a fatiga e o fallo muscular poden ter un efecto importante na súa aparición. Para iso, deseñar protocolos que permitan comparar configuracións da serie máis preto ou lonxe do fallo muscular, así como chegando ou sin chegar ata o fallo muscular, mentres manteñen igualados o resto de parámetros da carga, é de crucial importancia para elucidar esta cuestión. As series longas, próximas ou ata o fallo muscular, provocan contracción musculares máis longas, un fluxo de sangue empeorado e unha maior fatiga que as configuracións da series curtas, lonxe da aparición do fallo muscular. Estos factores mencionados son contribuíntes na producción de histamina ou na activación dos receptores de histamina, un importante determinante do efecto hipotensivo postexercicio. A activación dos receptores de histamina proporcionan protección contra a fatiga durante o exercicio e xogan un rol importante durante a recuperación, polo que é plausible que as configuracións da serie longas poidan axudar a aparición do efecto hipotensivo postexercicio. Ademáis, a interacción co tipo de exercicio pode ser de interese xa que a inclusión de membros inferiores tense amosado como un importante modulador do efecto hipotensivo.

En relación ao esforzo percibido durante o exercicio, esta tese proponse estudar por primeira vez o efecto da configuración da serie na percepción de esforzo. Ademáis, este estudo pode contribuír a elucidar como outros parámetros da carga inflúen na percepción de esforzo, xa que mentres a duración da serie é diferente en cada configuración, a relación entre o traballo e a pausa, a intensidade da carga e o volume total realizado están igualados. Previamente intentouse resolver esta cuestión cun protocolo que igualaba a relación entre o traballo e a pausa, sin ter en conta o número total de repeticións que se podían realizar. Con este deseño, tendo en conta o número total de repeticións realizable, esta eiva elimínase. Ademáis, estudarase a súa interacción co tipo de exercicio, co fin de obter un coñecemento máis profundo da configuración da serie, contribuíndo así a convertir as escalas de esforzo percibido en ferramentas útiles para monitorizar a percepción de esforzo durante o exercicio de forza.

Os obxectivos e hipóteses prantexados nos tres estudos realizados nesta tese son:

Estudo I: A shorter set reduces the loss of cardiac autonomic and baroreflex control after resistance exercise.

O obxectivo foi analizar o efecto de distintas configuracións da serie submáximas dun exercicio de forza (*leg press*) no control cardíaco parasimpático autónomo e barorreflexo e na presión arterial postexercicio. Hipotetizouse que as configuracións da serie máis longas sen chegar ata o fallo muscular producirán unha perda maior no control cardíaco e unha maior redución da presión arterial despois do exercicio en comparación coas configuracións da serie máis curtas.

Estudo II: Exercise type affects cardiac vagal autonomic recovery after a resistance training session.

O obxectivo foi analizar o efecto dunha configuración da serie máxima en comparación cunha configuración da serie submáxima cun deseño de descanso entre repeticións no control cardíaco vagal e na presión arterial postexercicio e a súa interacción con dous tipos de exercicio con diferente cantidade de masa muscular implicada (*bench press* e *parallel squat*). Hipotetizouse que a configuración da serie máxima e o exercicio con máis masa muscular implicada (*parallel squat*) producirán unha maior retirada vagal cardíaca e un maior efecto hipotensivo en comparación cunha configuración da serie submáxima cun deseño de descanso entre repeticións e co exercicio con menos masa muscular implicada *(bench press*).

Estudo III: Effects of set configuration of resistance exercise on perceived exertion.

O obxectivo foi analizar o efecto dunha configuración da serie máxima en comparación cunha serie submáxima cun deseño de descanso entre repeticións no esforzo percibido e a súa interacción con dous tipos de exercicio con diferente cantidade de masa muscular implicada (*bench press* e *parallel squat*). Hipotetizouse que a configuración da serie máxima e que o exercicio con máis masa muscular (*parallel squat*) producirán un maior esforzo percibido en comparación cunha configuración da serie curta cun deseño de descanso entre repeticións e co exercicio con menos masa muscular implicada (*bench press*).

Os principais achados da tese foron que: a) as configuracións da serie longas máximas e submáximas produciron unha redución maior da actividade parasimpática cardíaca, como foi observado pola perda de control autónomo e barorreflexo, en comparación con configuracións da serie curtas. b) unha configuración da serie curta cun deseño de descanso entre repeticións preservou o control parasimpático do corazón, como foi observado polas reducións non significativas no control autónomo e barorreflexo despois do exercicio. c) o efecto da configuración da serie no control autónomo vagal está afectado polo tipo de exercicio realizado durante un deseño con descanso entre repeticións, cun maior mantemento da actividade parasimpática no exercicio con menos masa muscular implicada (*bench press*), en comparación co exercicio con máis masa muscular implicada (*parallel squat*), mais non durante as configuracións da serie máximas. d) as configuracións da serie máximas, mais non as submáximas, producen a aparición do efecto hipotensivo postexercicio nunha sesión cun volume total reducido. e) o efecto da configuración da serie no efecto hipotensivo postexercicio non está modulado polo tipo de exercicio realizado nunha sesión con volume total reducido. f) unha configuración da serie máxima produce un esforzo percibido maior en comparación cunha configuración da serie curta como o deseño con descanso entre repeticións, indicando que a configuración da serie determina o esforzo percibido. g) o efecto da configuración da serie no esforzo percibido foi dependente do exercicio realizado, con diferenzas observadas entre os protocolos con menos masa muscular implicada (*bench press*) mais non entre os protocolos con máis masa muscular implicada (*parallel squat*).

Os resultados dos estudos realizados mostraron que cando se igualan a intensidade de carga, o volume total realizado e a relación entre o traballo e a pausa, a configuración da serie determina a perda de control parasimpático autonómo e barorreflexo, independentemente da comparación entre protocolos submáximos ou entre protocolos submáximos e máximos.

O maior candidato para a perda do control autónomo cardíaco de orixe vagal é a participación glicolítica da sesión, con maiores redución na actividade parasimpática con maiores produccións de lactato. Neste sentido, observaronse relacións entre o control parasimpático cardíaco e a participación glicolítica tanto despois de inxeccións de lactato como despois da realización de exercicios de forza. Aínda que a producción de lactato non se analizou neste estudo, previamente amosáronse diferenzas na participación glicolítica entre configuracións da serie curtas e longas despois dunha sesión de adestramento de forza. Ademáis, a producción de lactato correlaciona fortemente co rendemento mecánico, unha variable que foi maior nas configuracións da serie curtas. As configuracións da serie curtas cun deseño con descanso entre repeticións produciron unha redución non significativa da actividade vagal, posiblemente debido a elevacións non signficativas da producción de lactato. As diferenzas entre deseños son posiblemente debidas a unha rexeneración parcial da fosfocreatina nas configuracións da serie curtas, permitindo unha menor demanda da glicólese anaeróbica. Cando se ten en conta o tipo de exercicio realizado no deseño de configuración de serie curta con descanso entre repeticións, non se observan reducións cando se realizaron os exercicios *leg press* ou *bench press*, mais si cando se realizou *parallel squat.* Aínda que neste momento trazar inferencias que xustifiquen estas diferenzas pode levar a conclusións erróneas, semella que a habilidade das configuracións da serie curtas como o deseño con descanso entre repeticións para reducir a perda de control vagal cardíaco é dependente do exercicio realizado.

Por outra banda, o efecto dos diferentes tipos de exercicio realizados no control autónomo vagal tamén depende das configuracións da serie empregadas. Neste sentido, os nosos resultados mostran diferenzas entre exercicios cando foron realizados con configuracións curtas, mais non se atoparon diferenzas cando os exercicios se realizaron ata o fallo muscular. Isto suxire que procesos fisiolóxicos como os que acontecen durante o fallo muscular poden afectar ao control autónomo cardíaco, relegando a un segundo plano o efecto da implicación glicolítica.

A configuración da serie tamén afectou o control parasimpático cardíaco de orixe reflexo, con maiores reducións na sensibilidade barorreflexa con configuracións da serie longas en comparación cun deseño con descanso entre repeticións. O noso estudo é o primeiro en mostrar como un só exercicio de forza é capaz de afectar ao control barorreflexo. Así mesmo, cunha configuración da serie curta como o deseño con descanso entre repeticións, é posible realizar unha sesión de forza sen afectar ao control cardíaco barorreflexo. O control na redución da actividade parasimpática cardíaca mediante a configuración da serie ten aplicacións prácticas en individuos con risco cardiovascular, xa que a perda de control parasimpático cardíaco postexercicio ten de ser interpretada como un efecto perxudicial transitorio xa que 30 minutos despois dun exercicio existe un risco maior de sufrir un avento cardíaco como consecuencia desta perda na actividade vagal.

A configuración da serie tamén determinou a hipotensión postexercicio. Os resultados amosaron que cando a configuración é máxima, é dicir, ata o fallo muscular, produce unha redución da presión arterial sistólica e diastólica en comparación cunha sesión control. Pola contra, sesións submáximas tanto longas como curtas non produciron hipotensión postexercicio. Por outra lado, a interacción entre a configuración da serie e o tipo de exercicio (é decir, *bench press* ou *parallel squat*) non produciu diferenzas. Esta falta de diferenzas entre tipos de exercicio chegando ata o fallo muscular é consistente coa literatura, suxerindo que o efecto da masa muscular é relevante con volúmenes maiores e que seguramente sexa manos importante coa aparición do fallo muscular. Estudos previos reportaron que un mínimo volume é necesario, incluso coa aparición do fallo muscular, para a aparición da hipotensión postexercicio despois do exercicio de forza. No nosos estudos, cunha configuración da serie ata o fallo muscular e cunha redución progresiva do número de repeticións en cada nova serie como consecuencia da fatiga muscular, observouse o efecto hipotensivo cun volume total excesivamente baixo en comparación con estudos anteriores.

Isto desafía en parte a premisa de que un volume total elevado é crucial na aparición do efecto hipotensivo. Sen embargo, non é contradictorio co anteriormente reportado, xa que ambas variables pódense deber ao mesmo efecto fisiolóxico. Neste sentido, un dos mecanismos obligatorios na aparición da hipotensión postexercicio, a vasodilatación dependente da activación dos receptores histaminérxicos, pode que aconteza tanto chegando ao fallo muscular como realizando grandes volumes. Tendo isto en conta, pode que o fallo muscular intencionado e deliberado da configuración da serie máxima utilizada poda desprazar a importancia do volume total realizado a un rol secundario. Esta redución na presión arterial postexercicio observada nos deseños ata o fallo muscular ten unha aplicación práctica como estratexia non farmacolóxica para o tratamento ou a prevención da hipertensión, xa que os efectos agudos postexercicio observados contribúen ás reducións crónicas da tensión arterial.

Por último, a configuración da serie tamén determina o esforzo percibido. Os resultados indican que cando a intensidade da carga, o volume e a relación entre o traballo e a pausa están igualados, unha configuración da serie máxima produce un esforzo percibido maior que unha configuración da serie curta cun deseño de descanso entre repeticións. Así, os resultados obtidos apoian investigacións previas que argumentan que a duración das repeticións e da serie median a percepción de esforzo. Deste xeito, a aparición do fallo muscular é o gran determinante do esforzo percibido, xa que a duración das repeticións e da serie son lóxicamente máis extensas con cada configuración da serie máis longa. En relación a interacción entre a configuración da serie e o tipo de exercicio, para una mesma configuración da serie observáronse diferenzas no exercicio con menos masa muscular implicada (*bench press),* con valores máis altos na configuración da serie máis longa, pero non se atoparon diferenzas no exercicio con máis masa muscular implicada (*parallel squat*). Os mecanismos fisiolóxicos que explican esas diferenzas son descoñecidos, sen embargo, os resultados obtidos reflexan as diferenzas obtidas no rendemento mecánico.

Neste sentido, aínda que as correlacións entre os valores de esforzo percibido e diferentes parámetros fisiolóxicos son difíciles de atopar, o esforzo percibido foi previamente capaz de reflexar as diferenzas na producción de lactato ou na actividade muscular, ambos indicadores de fatiga neuromuscular. Da mesma maneira, as diferenzas entre exercicios foron observadas para a configuración da serie cun deseño con descanso entre repeticións, mais non na configuración da serie máxima. Na configuración da serie curta, o exercicio con máis masa muscular implicada *(parallel squat*) produciu maiores valores que o exercicio de menos masa muscular (*bench press*). Unha maior producción de lactato e un porcentaxe maior en relación a máxima manifestación de forza poden ter mediado nos resultados obtidos para esa configuración da serie. Pola contra, na configuración da serie máxima non se atoparon diferenzas entre exercicios. Unha serie ata o fallo muscular ten que dar, por definición, valores máximos de esforzo percibido, o que xustifica a falta de diferenzas observadas entre ambos. O coñecemento de como a configuración da serie e outros parámetros da carga determinan e modulan o esforzo percibido poderá axudar a controlar a carga externa durante a sesión, o que permitirá unha maior adherencia ao exercicio así como evitar procesos de sobreadestramento e enfermidade coa axuda de outras ferramentas do control da carga.

Os principais conclusións deste tese son:

- Un exercicio de forza con configuracións da serie longas produce unha retirada vagal cardíaca e unha perda de sensibilidade barroreflexa maior en comparación con configuracións da serie curtas.
- Unha configuración da serie curta cun deseño con descanso entre repeticións produce unha redución non significativa da actividade parasimpática, observada por valores comparables na actividade vagal cardíaca e na sensibilidade barorreflexa.

- O efecto da configuración da serie é modulado polo tipo de exercicio realizado, con maiores reducións no control vagal cardíaco nas configuracións da serie longas en comparación coas curtas para *bench press*, mais non para *parallel squat.*
- O efecto do tipo de exercicio é modulado pola configuración da serie utilizada, con grandes reducións no control vagal cardíaco no exercicio con máis masa muscular implicada (*parallel squat*) en comparación co exercicio con menos masa muscular implicada (*bench press*) realizando unha configuración da serie curta, mais non realizando series ata o fallo.
- A diferenza dos exercicios *leg press* e *bench press*, unha configuración curta con descanso entre repeticións realizando *parallel squat* produce reducións no control cardíaco, suxerindo que a aplicabilidade das configuracións curtas depende do exercicio realizado.
- Un só exercicio de forza é suficiente para producir unha redución na actividade vagal cardíaca e na sensibilidade barorreflexa despois dunha sesión.
- Un exercicio de forza cun volumen pequeno realizado con configuracións da serie submáximas, curtas ou longas, non produce hipotensión postexercicio, mentres que realizándoo cunha configuración longa, ata o fallo muscular, si que a produce.
- Un exercicio de forza realizado cun volumen pequeno produce un efecto hipotensivo comparable e independente do tipo de exercicio realizado.
- Un exercicio de forza cunha configuración da serie longa produce un esforzo percibido maior en comparación cunha configuración da serie curta.
- O efecto da configuración da serie está modulado polo tipo de exercicio empregado, con maiores valores de esforzo percibido na configuración da serie longa en comparación coa curta cando se realiza *bench press*, mais non cando se realiza *parallel squat*.
- O efecto do tipo de exercicio realizado está modulado pola configuración da serie empregada, con valores máis altos de esforzo percibido no exercicio con máis masa muscular implicada (*parallel squat*) en comparación co exercicio con menos masa muscular implicada (*bench press*) realizando unha configuración da serie curta, mais non realizando unha configuración da serie ata o fallo.

Appendix B

Informed consent

Anexo I

Información e consentimento informado.

Información ao participante

TÍTULO:

Effect of the set configuration of resistance exercise on cardiovascular control and perceived exertion: Interaction with the type of exercise.

INVESTIGADOR PRINCIPAL:

Este documento ten por obxecto ofrecerlle información sobre un estudo de investigación no que se lle invita a participar. Este estudo estase realizando dende a Facultade de Ciencias do Deporte e a Educación Física, Universidade da Coruña.

Se decide participar no mesmo, debe recibir información persoalizada do investigador, ler antes este documento e facer todas as preguntas que necesite para comprender os detalles sobre o mesmo. Si así o desexa, pode levar o documento, consultalo con outras persoas e tomarse o tempo que sexa necesario para decidir si participar ou non.

A participación neste estudo é completamente voluntaria. Podes decidir non participar, ou si desexas paricipar, cambiar de opinión retirando o consentimento informado en calquera momento sen obriga de dar explicacións.

CAL É O PROPÓSITO DO ESTUDO?

O obxectivo é coñecer o efecto cardiovascular agudo na configuración dos tres protocolos de forza coa mesma intensidade e carga total en dous tipos diferentes de exercicio. Para iso realizaranse unhas medicións antes, durante e despois que analizarán parámetros hemodinámicos e mecánicos.

POR QUE ME OFRECEN PARTICIPAR A MIN NESTE ESTUDO?

Porque cumpres cos criterios que se especifican no deseño da investigación de ser un suxeito san, adulto e non medicado e con coñecemento no adestramento de forza.

EN QUE CONSISTE A MIÑA PARTICIPACIÓN?

En 12 sesións, 3 formativas, 1 de toma de datos, 2 de control e 6 de avaliación relacionadas co obxecto de estudo. As sesións realizaránse polo menos con 72 horas entre elas.

Valoraranse diferentes variábeis como respostas cardiovasculares (frecuencia cardiaca, variabilidade da frecuencia cardiaca e presión arterial), mecánicas (velocidade de execución e tempo de traballo) y fisiopsicolóxicas (esforzo percibido).

Para garantir unhas condicións experimentais adecuadas deberase:

Realizar todas as probas na mesma franxa horaria segundo a dispoñibilidade individual.
Non tomar medicamentos de ningún tipo pola posibilidade de interferir nos resultados.
Non inxerir alimentos nas 3 horas anteriores nin cafeína no mesmo día.
Non realizar actividade física nas 24 horas anteriores.
Levar roupa e calzado apropiados.

É necesario que se decide participar neste estudo, comprométase a asistir as sesións de toma de datos. No momento en que a falta de asistencia sexa repetida e provoque que non se cumplan os periodos de tempo fixados, decidirase apartalo do estudo.

QUE RISCOS OU INCONVINTES TEN?

A realización das distintas sesións producirán fatiga e maniotas. Para reducir cualquer risco de lesión, todas as valoracións irán precedidas por un quecemento específico deseñado e dirixido por un especialista. As execucións dos exercicios serán supervisadas polo menos por dous investigadores, que prestarán a axuda necesaria ao deportista. No periodo formativo realizaranse as correccións e recomendacións oportunas para minimizar os riscos.

Se durante o transcurso do estudo se coñecera información relevante que afecte a relación entre o risco e o beneficio da participación, se lle transmitirá para que poida decidir abandoar ou continuar.

OBTEREI ALGÚN BENEFICIO POR PARTICIPAR?

Non obterá ningún beneficio, máis aló da información recollida se desexa coñecela. Aprenderá certos aspectos do adestramento e avaliación polos que mostre interés.

RECIBIREI A INFORMACIÓN QUE SE OBTEÑA DO ESTUDO?

Facilitarase un resumo cos resultados do estudo e dos resultados das probas se así o solicita. Os resultados poden non tener unha aplicación clínica nin unha interpretación clara, polo que se quer dispoñer deles, deberían ser comentados có investigador principal do estudo.

PUBLICARANSE OS RESULTADOS DESTE ESTUDO?

Os resultados serán publicados en publicacións científicas para a súa difusión, pero non se transmitirá ningún dato que poida levar a identificación dos participantes.

COMO SE PROTEXERÁ A CONFIDENCIALIDADE DOS MEUS DATOS?

O tratamiento, comunicación e cesión dos seus datos farase conforme ao disposto pola Lei Orgánica 15/1999, do 13 de decembro, de protección de datos de carácter persoal. En todo momento, poderá acceder aos datos, correxilos ou cancelalos.

Só o equipo investigador terá acceso a todos os datos obtidos no estudo. Poderase transmitir a terceiros información que non sexa identificada. No caso de que algunha información sexa transmitida a outros paises, realizarase con un nivel de protección de datos equivalente, como mínimo, ao esixido pola normativa do noso país. A transmisión de datos a terceiros ten por finalidade realizar unha análise máis exhaustiva dalgúns parámetros rexistrados que por razóns técnicas non poidan ser analizadas no noso laboratorio.

EXISTEN INTERESES ECONÓMICOS NO ESTUDO?

Non será retribuido por participar.

É posíbel que os resultados dos estudos deriven en productos comerciais ou patentes. Neste caso, non participará nos beneficios económicos orixinados.

Todas as medicións levaranse a cabo nas instalacións da Facultade de Ciencias do Deporte e a Educación Física da Universidade da Coruña, polo que en ningún momento contemplarase o alquiler ou arrendamento de instalacións.

QUEN PODE DARME MÁIS INFORMACIÓN?

Pode contactar con Eliseo Iglesias Soler no teléfono 696462950 ou na dirección de correo eliseo@udc.es para máis información.

MOITAS GRAZAS POLA SÚA COLABORACIÓN

Consentimento informado

TÍTULO:

Effect of the set configuration of resistance exercise on cardiovascular control and perceived exertion: Interaction with the type of exercise.

Eu,

☐ Lín a folla de información ao participante do estudo anteriormente nomeado que se me entregou, falei co investigador principal e fíxenlle todas as preguntas sobre o estudo necesarias para comprender as condicións e considero que recibín suficiente información sobre o estudo.

☐ Comprendo que a miña participación é voluntaria, e que podo retirarme do estudo cando queira, sen ter que dar explicacións.

☐ Accedo a que se utilicen os meus datos nas condicións detalladas na folla de información ao participante.

☐ Presto libremente a miña conformidade para participar no estudo.

Respecto á conservación e utilización futura dos datos e/o mostras detalladas na folla de información ao participante:

☐ NON accedo a que os meus datos sexan conservados unha vez terminado o presente estudo.

☐ Accedo a que os meus datos consérvense unha vez terminado o estudo, sempre e cando non sexa posíbel, mesmo polos investigadores, identificalos por ningún medio.

☐ Accedo a que os datos e/ou mostras consérvense para usos posteriores en liñas de investigación relacionadas coa presente, e nas condiciones anteriormente sinaladas.

En canto aos resultados das probas realizadas:

☐ DESEXO coñecer os resultados das miñas probas. ☐ NO DESEXO coñecer os resultados das miñas probas.

O/a participante,	O/a investigador/a principal,
Fdo.:	Fdo.:
Data:	Data:

Appendix C

Publications that led to the thesis

Articles

1. Mayo X, Iglesias-Soler E, Carballeira-Fernández E, Fernández-Del-Olmo M. A shorter set reduces the loss of cardiac autonomic and baroreflex control after resistance exercise. Eur J Sport Sci. 2015 Nov 15;1391(November):1–9.

2. Mayo X, Iglesias-Soler E, Fariñas-Rodríguez J, Fernández-Del-Olmo M, Kingsley JD. Exercise type affects cardiac vagal autonomic recovery after a resistance training session. J Strength Cond Res. 2016 Jan 22 [Publish ahead-of-print].

3. Mayo X, Iglesias-Soler E, Fernández-Del-Olmo M. Effects of set configuration of resistance exercise on perceived exertion. Percept Mot Skills. 2014 Dec 2;119(3):825–37.

Conference Proceedings

1. Mayo X, Iglesias-soler E, Dopico-Calvo X. Cardiac and vascular autonomic modulation by different set configurations of resistance exercise. 19th ECSS Congress. Amsterdam; 2014.

2. Mayo X, Iglesas-Soler E, Dopico X, Fariñas-Rodríguez J. Effect of set configuration on blood pressure during resistance exercise. NSCA IV International Conference. Murcia; 2014.

3. Mayo X, Iglesias-Soler E, Fustes-Piñeiro S, González-Hernández R. The effect of set configuration and type of resistance exercise on recovery blood pressure. NSCA IV International Conference. Murcia; 2014.

4. Mayo X, Iglesias-soler E, Fustes-Piñeiro S, González-Hernández R. Neuromuscular performance is affected by set configuration and the type of resistance exercise. NSCA IV International Conference. Murcia; 2014.

5. Mayo X, Iglesias-Soler E, Carballeira E, Sánchez-Otero T, Castro-Gacio X. Perceived exertion during different set configurations in bench press and its relationship with blood pressure and power output. 18th ECSS Congress. Barcelona; 2013.

6. Mayo X, Iglesias-soler E, Sánchez-Otero T, Carballeira E. Estructura temporal, respuestas hemodinámicas, rendimiento mecánico y esfuerzo percibido en el ejercicio de fuerza. XII Congreso Internacional de la Asociación Española de Ciencias del Deporte. Granada; 2012.

7. Mayo X, Fariñas J, Giráldez-García M, Fernández-del-Olmo M, Iglesias-Soler E. Un ejercicio de fuerza afecta a la sincronización parasimpática entre la actividad autónoma y barorrefleja. IX Congreso Internacional de la Asociación Española de Ciencias del Deporte. Toledo; 2016.