Ischemic preconditioning improves autonomic modulation after session of resistance exercise

Pré-condicionamento isquêmico melhora a modulação autonômica após sessão de exercício resistido

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ABSTRACT
The aim of this study was to investigate the acute effect of ischemic preconditioning (IPC) in a session of resistance exercise (RE) for upper and lower limbs on the heart rate variability (HRV) in normotensive and trained men. sixteen normotensive and trained men visit the laboratory in five sessions in non-consecutive days. The first two sessions subjects performed one repetition maximum (RM) test and retest, and the next three visits they performed the experimental protocols: a) RE (CON), b) IPC+RE (IPC), c) SHAM+RE (SHAM). RE were performed in 3 sets at 80% 1RM until concentric failure. IPC consisted of 4x5-mins of vascular occlusion at 220 mmHg alternating with 5-min of reperfusion. SHAM protocol followed the same IPC method with 20mmHg vascular occlusion. A significant decrease in LFnu and RMSSDms ($p=0.001$) was found from baseline for IPC, SHAM, and CON. A significant increase in HFnu and LF/HF ($p=0.001$) was found from baseline for IPC, SHAM, and CON. A significant decrease in LFnu and LF/HF was observed from 60-min post for IPC vs. SHAM and IPC vs. CON ($p<0.05$). A significant increase in HFnu was observed from 60-min post for IPC vs. SHAM and IPC vs. CON ($p<0.05$). A significant increase in RMSSDms was found from post-60 for IPC vs. SHAM ($p < 0.05$). RE followed IPC shows significantly improvements in the autonomic cardiac modulation, accelerating the autonomic recovery after the RE session, by increasing the vagal activity and reducing the sympathetic activation when compared to RE and SHAM protocols.

Key-words: ischemic preconditioning, autonomic response, heart rate variability, resistance exercise, vascular occlusion.

RESUMO
O objetivo do presente estudo foi investigar o efeito agudo do pré-condicionamento isquêmico (PCI) em uma sessão de exercício resistido (ER) para membros superiores e inferiores sobre a variabilidade da frequência cardíaca (VFC) em homens normotensos e treinados. O estudo foi composto por dezesseis homens normotensos e treinados. Consistiu em cinco sessões no laboratório em dias não consecutivos. Nas duas primeiras sessões, os sujeitos realizaram um teste e reteste de uma repetição máxima (RM), e nas três visitas seguintes realizaram os protocolos experimentais: a) RE (CON), b) PCI + RE (PCI), c) SHAM + RE (SHAM) Os ER foram realizados em 3 séries a 80% 1RM até a falha concêntrica. O IPC consistiu em 4x5 minutos de oclusão vascular a 220 mmHg alternando com 5 minutos de reperfusão. O protocolo SHAM seguiu o mesmo método do IPC com oclusão vascular de 20 mmHg. Uma diminuição significativa em

LF¬nu e RMSSDms (p = 0,001) foi encontrada na linha de base para PCI, SHAM e CON. Um aumento significativo em HFnu e LF / HF (p = 0,001) foi encontrado na linha de base para PCI, SHAM e CON. Uma diminuição significativa em LF¬nu e LF / HF foi observada a partir de 60 minutos pós para IPC vs. SHAM e IPC vs. CON (p <0,05). Um aumento significativo no HFnu foi observado a partir de 60 minutos pós para PCI vs. SHAM e PCI vs. CON (p <0,05). Um aumento significativo no RMSSDms foi encontrado a partir da pós-60 para PCI vs. SHAM (p <0,05). O ER seguido do PCI mostra melhora significativa na modulação cardíaca autonômica, acelerando a recuperação autonômica após a sessão de ER, por aumentar a atividade vagal e reduzir a ativação simpática quando comparado aos protocolos ER e SHAM.

Palavras-chave: pré-condicionamento isquêmico, resposta autonômica, variabilidade da frequência cardíaca, exercício resitido, oclusão vascular

1 INTRODUCTION

Ischemic preconditioning (IPC) is a maneuver consisting of vascular occlusion, with alternating moments of reperfusion. Usually, the IPC is applied by a pneumatic tourniquet before performing the exercise in a non-invasive way (DA SILVA NOVAES et al., 2020). In the reperfusion phase, the application of IPC can increase blood flow in the skeletal muscles (WANG et al., 2004), in the liver (KANORIA et al., 2006), in the heart (ZHOU et al., 2007) and in the kidneys (ALI et al., 2007).

The physiological mechanisms involved in the IPC effect are based on the activation of adenosine receptors and nitric oxide secretion that cause vasodilation after reperfusion, in the activation of the mitochondrial K+ATP channels responsible for hyperpolarization in the cardiac muscle cell, thus delaying the action potential. It also slows the consumption of ATP and PCr (adenosine triphosphate and phosphocreatine, respectively) energy by the muscle, increasing its tolerance during myocardial ischemia (PANG et al., 1995).

One of the first studies of IPC effect was carried out by Murry et al. (1996), which investigated the effect of IPC on 40-min sustained ischemia in the circumflex coronary artery of dog hearts. The results indicated that the group that performed IPC before undergoing 40-minute ischemia had an infarct area limited to 25% of that observed in the control group. These data demonstrate the fact that brief moments of ischemia, and reperfusion, provide an important cardioprotective effect.

Based on the hypothesis that IPC may increase heart blood flow and post-reperfusion muscle performance, skeletal muscle research has been initiated by associating IPC with physical exercise (DE GROOT et al., 2010). Several studies have been examined the effect of IPC on sports performance (DE GROOT et al., 2010;

LISBÔA et al., 2017; WILLIANS et al., 2018) and on resistance exercises (RE) (DA SILVA NOVAES et al., 2020; TELLES et al., 2020; PANZA et al., 2019).

An acute bout of RE is capable of changing the cardiac autonomic modulation, specifically causing the inhibition of the vagal nerve parasympathetic nervous system (PSNS) and an increase in the activity of the sympathetic nervous system (SNS) (KINGSLEY; FIGUEROA, 2016). Thus, methods that improve cardiac autonomic modulation could attenuate the acute effect of RE on the cardiovascular are pertinent and necessary. In this sense, IPC has the potential to induce powerful protection against injury caused by prolonged ischemia and subsequent reperfusion at a remote location, for example in the heart (KHARBANDA et al., 2002). The vagal branch of the autonomic nervous system seems to play a central role in IPC-mediated protection (MASTISTSKAYA et al., 2012). Besides that, the IPC not only protects against injury but may improve some cardiovascular health markers (BARROS et al., 2020; COCKING et al., 2017; JEFFRIES et al., 2018) and increase PSNS activity in healthy men (ENKO et al., 2011), which is cardioprotective.

Therefore, evidence of ischemia-reperfusion models in resting healthy men maintains that IPC is an effective intervention in the clinical area, capable of causing local and systemic cardioprotective effects through the improvement of cardiac and hemodynamic autonomic system activity (PANZA et al., 2019; ROSENBERG et al., 2018; BILLAH et al., 2019). However, it has not yet been verified whether IPC can improve autonomic modulation after acute RE. Thus, the aim of the study was to investigate the acute effect of ischemic preconditioning in a session of resistance exercise for upper and lower limbs on the heart rate variability in normotensive and trained men. We hypothesized that applying IPC before the RE session will improve autonomic modulation in comparison to other experimental protocols.

2 METHODS

2.1 PARTICIPANTS

The study included 16 normotensive men (Age: 25.3 ± 1.7 years, Weight: 78.4 ± 6.2 kg, Height: 176.9 ± 5.4 cm, Body Mass Index: 25.1 ± 1.5) with experience on RE for at least one year (5.0 ± 1.6 yrs). The sample size was performed using G*Power 3 software. Based on a previous analysis, an n of 16 subjects was calculated after using a power of 0.80, $\alpha = 0.05$, a correlation coefficient of 0.5, the Nonsphericity correction of 1 and an effect size of 0.32. It was verified that the sample size was sufficient to provide

83.8% of the statistical power. For the calculation of the sample, the procedures suggested by Beck (2013) were adopted. Subjects were excluded from the study if respond positively to any of the items in the Physical Activity Readiness Questionnaire (SHEPHARD, 1988) or missed one of the sessions of the collection procedures in the laboratory or presented some type of osteomioarticular lesion in the upper or lower limbs, obesity, hypertension, users of supplements, medications and smokers. After explaining the risks and benefits of the research, subjects signed the informed consent form, elaborated according to the Helsinki Declaration. The study complied with Resolution 466/12 of the National Health Council and was approved by the local Ethics and Research Committee of Volta Redonda University Center under protocol number 2.699.294.

2.2 EXPERIMENTAL DESIGN

The present study was performed over five visits on non-consecutive days (3-day interval), always at the same time of day (±1 hour) to avoid circadian influence. During the first visit to the laboratory the informed consent form was signed. Then, the Physical Activity Readiness Questionnaire was answered and, immediately after, the anthropometric data was assessed and the 1 repetition maximum (1RM) test was performed. In the second visit, a 1RM test was performed again to test the load reproducibility, which could not present a difference higher than 5% of the load found on the first test day. From the third to the fifth visit to the laboratory, the subjects were randomly assigned into one of the following experimental protocols: a) RE session at 80% of 1RM session protocol (CON); b) IPC+RE at 80% of 1RM (IPC); c) SHAM+RE at 80% of 1RM (SHAM). The experimental design of the study can be observed in figure 1. RE session was composed of six exercises for upper and lower limbs: bench press (BP), leg press 45 ° (LP), lat pulldown (LPD), hack machine squat (HM), shoulder press (SP), Smith back squat (SS). Each exercise was performed with a volume of three sets, at 80% of 1RM, until concentric failure, with 1-min and 30-sec of rest between sets, and 2-min between exercises.

2.3 PROTOCOLS

2.3.1 Ischemic Preconditioning

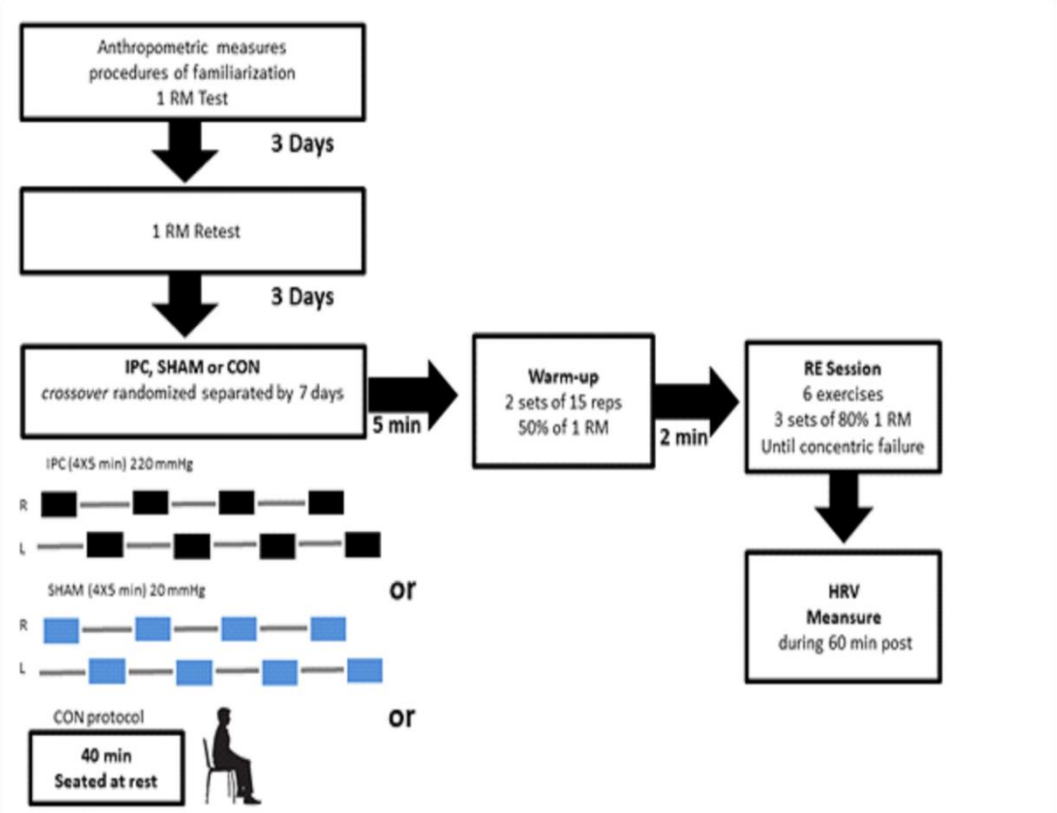
IPC protocol was composed of 4 cycles of 5-min of occlusion at 220 mmHg of pressure using a 57 cm x 9 cm pneumatic tourniquet applied around the subaxillary

region of the upper arm (komprimeterRiester®, Jungingen, Germany). Following each IPC protocol cycle there were 5-min of reperfusion at 0 mmHg. This resulted in a total intervention of 40-min. The pressure used, and the width of the cuff, are in agreement with a previous study (DA SILVA NOVAES et al., 2020; TELLES et al., 2020; TELLES et al., 2021). In order to certify that subjects had the blood flow obstructed during the intervention; the radial pulse was manually verified by digital palpation.

2.3.2 Sham

SHAM protocol session consisted of 4 cycles of 5-min of occlusion at 20 mmHg of pressure, as proposed in previous studies (DA SILVA NOVAES et al., 2020; TELLES et al., 2020). with 5-min of reperfusion at 0 mmHg for a total of 40-min. Subjects remained seated during the intervention, which lasted 40-min. The IPC and SHAM protocols can be seen in figure 1.

Figure 1. Experimental design. 1RM = one repetition maximum; IPC = ischemic preconditioning; SHAM = false protocol; RE = resistance exercise; CON = control protocol; HRV = heart rate variability.



2.3.3 Anthropometric assessment

Stature and body mass were measured with an accuracy of 0.5 cm and 0.1 kg, respectively. A stadiometer and a Filizola® brand balance beam scale was used, and all measurements were taken following the American College of Sports Medicine (2011) recommendations. These measures were later used to obtain the body mass index.

2.3.4 One repetition maximum test

The training load prescription was evaluated through the 1RM test (ACSM, 2011). The evaluations were carried out in the 1st and 2nd visits to the laboratory. The exercises were carried out bilaterally: BP, LP, LPD, HM, SP, and SS. The subjects used a 10-min interval for recovery time between exercises. For the warm-up, each subject performed two sets of 5-10 repetitions at 40-60% (1-min interval between sets), respectively, of the maximum perception of the individual's strength. After a 1-min rest interval, the third set was completed between 3-5 repetitions at 60-80% of the maximum perceived strength. After a rest interval (1-min), the force evaluation was started, in which up to 5 attempts were performed, adjusting the load before each new attempt. The recovery duration between trials was standardized at 3-5 min. The test was interrupted when the subject failed to perform the movement correctly. If the subject failed to perform the movement correctly, the maximum load recorded was the repetition that had proper execution.

2.3.5 Heart Rate Variability

An HR polar® RS 800CX monitor (Polar Electro Oy, Kempele, Finland) was used for 10-min of rest prior to each experimental protocol, immediately after the experimental protocol and every 10-min for up to 1-hour after each experimental protocol, with the subjects sitting during all measurements. All recordings were performed at the same time of day to avoid interferences of circadian rhythm on heart rate variability (HRV). The cardiofrequencimeter had a sampling frequency of 1000 Hz, fixed by an elastic band at the sternum (lower 1/3 portion) with simultaneous transmission to the watch fixed on the left wrist, where the record was stored. After, through an infrared sensor serial port, the data was transported and stored in the Polar Precision Performance program on an Acer® brand computer. These data were exported through a txt file for analysis via Kubios HRV Analysis Program 2.0 software (version 2.2, Kuopio, Finland). Thus, after noise removing by visual inspection of the tachogram

the most stable period of the signal was selected with a sampling frequency of 5 min, as recommended by the Task Force (1996). For spectral analysis of HRV, the time series of the R-R intervals underwent Fourier transform, in which the following frequency domain indexes were selected: Normalized (nu) low frequency (LFnu) of 0.04 to 0.15 Hz corresponding to sympathetic activity, high frequency (HFnu) from 0.15 to 0.40 Hz corresponding to parasympathetic activity, and the LF/HF ratio which corresponds with sympathovagal balance. In the time domain, the square root of the square sum of the differences between the R-R intervals divided by the number of R-R intervals (RMSSDms index) was selected, which corresponds to parasympathetic activity and was expressed in milliseconds (TASK FORCE, 1996; CAMPOS et al., 2018).

2.3.6 Statistical analyses

The results are presented as mean ± standard deviation. To test the normality of the data, the Shapiro-Wilk test was applied. In addition, we utilized the Levene test to examine the homoscedasticity, and the intraclass correlation coefficient (ICC) to evaluate the measures of reproducibility of the test and retest of the 1RM (ICC: BP = 0.98, LP = 0.99, LPD = 0.98, HM = 0.97, SP = 0.98, SS = 0.97). A 3x7 two-way repeated measures ANOVA was performed to determine the differences in the experimental protocols on the repeated factor of time (Baseline and 60-min post intervention) on the dependent variables (LFn.u, HFn.u, LF/HF ratio, and RMSSDms). A degrees of freedom of ANOVA values (df) was reported between and within groups and calculated using the formula F(A,B) = X,p =Y, Z%, where AB is the degrees of freedom, X is the ANOVA F value, Y is the p values and Z is the effect size. To determine the specific differences, the Bonferroni test post hoc was performed. Eta-squared (eta2) was reported as a measure of effect size for significant main effects and main interactions within the ANOVA. In addition, effect size estimates were calculated using the standardized mean difference to determine the magnitude of the treatment effects. The effect sizes represent the standardized within-group changes for each measurement time point compared with resting values (ES = [Mean Post 2 Mean Pre]/SD of the resting or pre-value). The magnitude of the ES was interpreted using the scale proposed by Rhea (2004) for recreationally trained subjects, where ,0.5, 0.50–1.25, 1.25–1.9, and 2.0 represented trivial, small, moderate, and large effects, respectively. Statistical analyses were performed using the statistical software package SPSS 21® (SPSS Inc., Armonk, NY, USA), adopting a critical level of significance of $p < 0.05$.

2.3.7 Results

All variables tested demonstrated a normal distribution ($p<0.05$). The effect size, p-values, and percentage changes (Δ%) for experimental conditions for LFnu, HFnu for each condition and time point are presented in (Table 1).

Table 1. IPC: ischemic preconditioning protocol; SHAM: False protocol; CON: Control protocol; ES = effect size; Δ% = difference between post and baseline moments in percentage; LF = low frequency in normalized units; HF = high frequency in normalized units;

	IPC			SHAM			CON		
	p	ES	Δ%	p	ES	Δ%	p	ES	Δ%	p
LF-n.u.										
Post 10	1.00	Moderate 1.77	31.14	0.01	Large 2.32	33.07	0.01	Large 2.57	43.88	0.01
Post 20	1.00	Moderate 1.57	27.67	0.01	Moderate 1.36	19.44	0.01	Large 2.22	37.86	0.01
Post 30	1.00	Moderate 1.69	29.65	0.01	Moderate 1.77	25.21	0.01	Large 2.25	38.32	0.01
Post 40	1.00	Moderate 1.35	23.72	0.01	Moderate 1.86	26.60	0.01	Large 2.08	35.45	0.01
Post 50	1.00	Moderate 0.98	17.28	0.02	Moderate 1.30	18.56	0.03	Moderate 1.58	27.00	0.01
Post 60	0.01	Small 0.56	9.78	0.85	Moderate 1.92	27.38	0.01	Moderate 1.72	29.40	0.01
HF-n.u.										
Post 10	1.00	Large -2.25	-56.19	0.01	Large -2.01	-65.63	0.01	Large -2.58	-64.99	0.01
Post 20	1.00	Large -2.03	-50.66	0.01	Moderate -1.28	-41.91	0.01	Large -2.31	-55.91	0.01
Post 30	1.00	Large -2.03	-50.66	0.01	Large -2.22	-55.29	0.01	Moderate -1.69	-55.91	0.01
Post 40	1.00	Moderate -1.78	-44.37	0.01	Moderate -1.66	-54.38	0.01	Large -2.04	-51.33	0.01
Post 50	1.00	Moderate -1.36	-34.10	0.07	Moderate -1.27	-41.55	0.01	Moderate -1.59	-40.03	0.01
Post 60	0.01	Small -0.89	-22.16	0.24	Moderate -1.70	-55.74	0.01	Moderate -1.81	-45.64	0.01

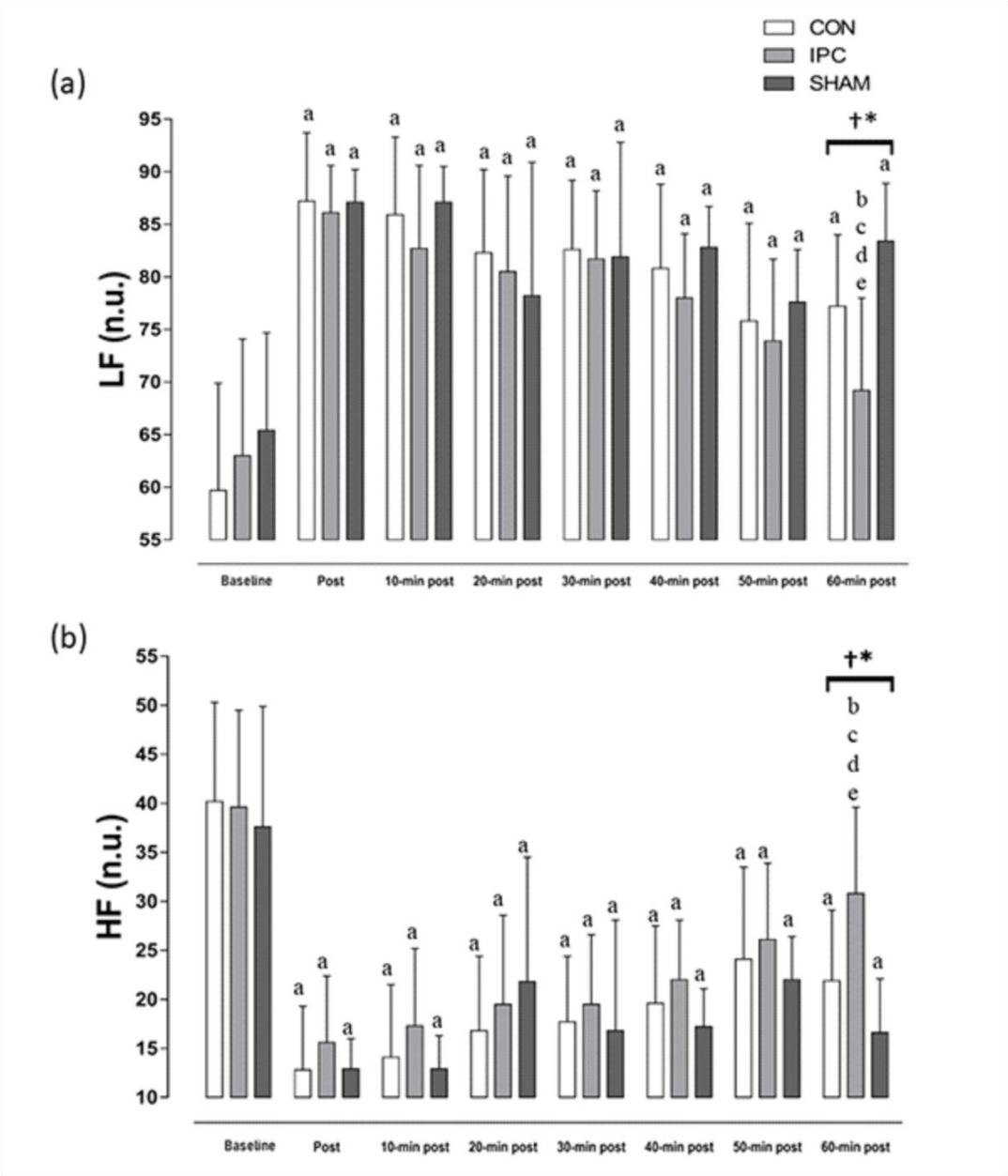
The effect size, p-values, and percentage changes (Δ%) for experimental conditions for LF/HF ratio, and RMSSDms for each condition and time point are presented in (Table 2).

Table 2. IPC: ischemic preconditioning protocol; SHAM: False protocol; CON: Control protocol; ES = effect size; Δ% = difference between post and baseline moments in percentage; LF/HF (ms2) ratio which corresponds with sympathovagal balance; RMSSD = standardized deviation of differences between adjacent normal R-R intervals

	IPC				SHAM			CON		
	P	ES	Δ%	p	ES	Δ%	P	ES	Δ%	p
LF/HF										
Post 10	1.00	Large 5.35	230.44	0.03	Large 9.84	303.43	0.01	Large 12.64	258.96	0.01
Post 20	1.00	Large 4.70	202.59	0.13	Large 7.44	171.75	0.27	Large 5.57	258.96	0.02
Post 30	1.00	Large 4.73	203.64	0.13	Large 10.40	320.77	0.01	Large 8.03	279.34	0.03
Post 40	1.00	Large 2.98	128.46	0.89	Large 6.33	195.37	0.09	Large 6.35	221.03	0.07
Post 50	1.00	Large 2.07	89.27	0.99	Large 3.36	103.68	0.97	Large 4.04	140.38	0.83
Post 60	0.01	Large 3.51	50.94	1.00	Large 10.16	213.53	0.03	Large 7.16	149.23	0.77
RMSSD.										
Post 10	1.00	Moderate -1.39	-70.42	0.01	Moderate -1.51	-69.18	0.01	Moderate -1.58	-76.69	0.01
Post 20	1.00	Small -1.19	-60.35	0.01	Moderate -1.46	-67.25	0.01	Moderate -1.43	-69.18	0.01
Post 30	1.00	Small -1.07	-54.49	0.01	Moderate -1.28	-58.69	0.01	Moderate -1.29	-62.35	0.01
Post 40	1.00	Small -0.83	-42.43	0.01	Small -1.21	-55.61	0.01	Small -1.20	-57.98	0.01
Post 50	1.00	Small -0.73	-37.29	0.01	Small -1.14	-52.30	0.01	Small -1.04	-50.21	0.01
Post 60	0.02	Small -0.68	-34.77	0.03	Small -1.07	-49.30	0.01	Small -0.95	-46.18	0.01

Significant differences were found in the comparison across the different experimental conditions for LFnu, HFnu, LF/HF ratio, and RMSSDms. A significant protocol x time interaction for LFnu ($F(1,15) = 12938.16$; $p= 0.001$; eta2 = 0.999) displayed decreases from baseline with IPC, SHAM and CON (Figure 2). A significant protocol x time interaction demonstrated increases compared with baseline for HFnu ($F(1,15) = 957.67$; $p = 0.001$; eta2 = 0.985) in IPC, SHAM and CON (Figure 2).

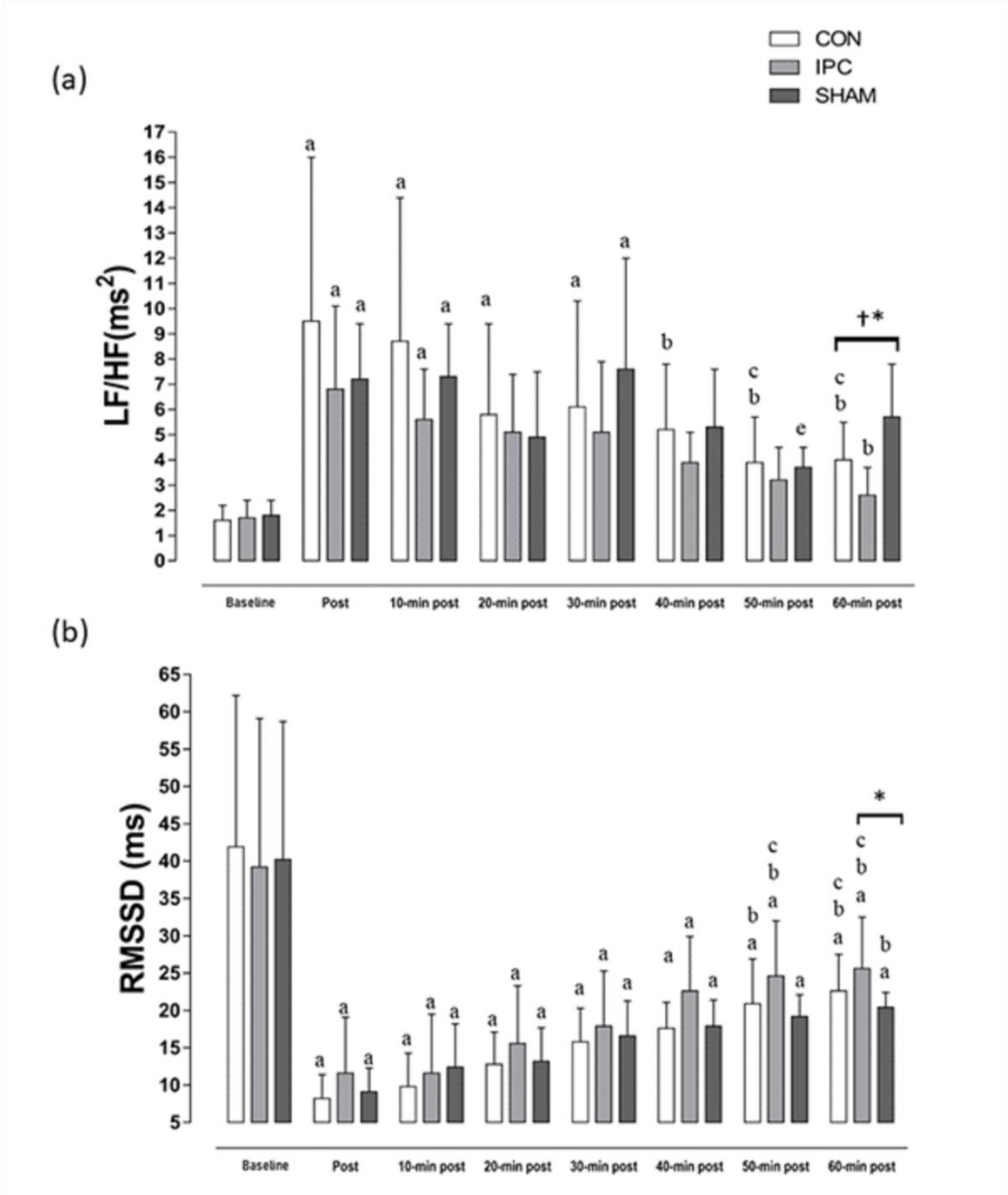
Figure 2. (a) LFnu response and (b) HFnu response post experimental protocol (mean ± sd). aSignificant difference compared to baseline; bSignificant difference compared to post; cSignificant difference compared to 10-min post; dSignificant difference compared to 20-min post; eSignificant difference compared to 30-min post; *Significant difference between IPC and SHAM; †Significant difference between IPC and CON. IPC = ischemic preconditioning; SHAM = false protocol; RE = resistance exercise; CON = control protocol.



A significant protocol x time interaction demonstrated increases compared with baseline for LF/HF ratio (F(1,15) = 328.25; p = 0.001; eta2 = 0.956) in IPC, SHAM and CON (Figure 3). Finally, there was a significant (interaction) decrease compared with baseline for RMSSD (F(1,15) = 837.38; p = 0.001; eta2 = 0.982) in IPC, SHAM and CON (Figure 3).

A significant difference ($p<0.05$) were observed between protocols for LF, HFnu (Figure 2) and LF/HF ratio with decreases from 60-min post with IPC vs. SHAM and IPC vs. CON (Figure 3). Between protocols for RMSSDms significant difference ($p<0.05$) were observed with increases from 60-min post with IPC vs. SHAM (Figure 3).

Figure 3. (a) LF/HF and (b) RMSSDms response post experimental protocol (mean ± sd). aSignificant difference compared to baseline; bSignificant difference compared to post; cSignificant difference compared to 10-min post; dSignificant difference compared to 20-min post; eSignificant difference compared to 30-min post; *Significant difference between IPC and SHAM; †Significant difference between IPC and CON. IPC = ischemic preconditioning; SHAM = false protocol; RE = resistance exercise; CON = control protocol.



3 DISCUSSION

This study was the first to investigate the effect of IPC applied before a resistance exercise session for upper and lower limbs on HRV in healthy young men. The main findings were that a) the IPC protocol presented lower SNS activity (LFnu) and higher PSNS activity (HFnu) at 1 hour (60-min post) when compared to immediate post-exercise, 10-min post, and 30-min post; b) that IPC significantly reduced SNS activity (LFnu) and sympathovagal balance (LF/HF ratio) and increased PSNS activity (HFnu) after the RE session when compared to SHAM and CON protocol 1 hour post exercise; and c) that the IPC protocol reduced sympathovagal balance from post to 20-min post without significant differences with pre or significant reductions immediately post exercise compared to 60-min post.

In our findings, in CON and SHAM protocols, a significant increase in LFnu was observed, along with a concomitant significant reduction in HFnu. Some studies of RE at different intensities corroborate the findings of our study. Rezk et al. (2006) verified the HRV response at two different intensities (40% 1RM and 80% 1RM), and Figueiredo et al. (2015a) investigated the intensities (60%, 70% and 80% 1RM) in the autonomic responses in untrained and trained men, respectively. In both studies, a significant increase of LFnu was observed, as well as a significant reduction of HFnu after a session of RE at all intensities, which demonstrates a greater activity of the SNS and a lower activity of the PSNS in response to acute RE.

In our study, the IPC protocol improved the autonomic modulation after the RE session, specifically increased PSNS activity and reduced SNS activity. These findings suggest that IPC, when coupled with RE, can accelerate autonomic recovery, unlike what happens when RE was utilized alone (as was the case in the CON). Other studies have shown that the higher volume of RE sets (FIGUEIREDO et al., 2015b), and a shorter interval between sets (FIGUEIREDO et al., 2015a; LEMOS et al., 2018) also showed a significant increase in SNS activity and a significant reduction in PSNS activity. Therefore, it appears that utilizing RE with high intensity, high volume, and short interval time are able to increase the activity of the SNS. Collectively, this represents higher cardiovascular stress (FIGUEIREDO et al., 2015a; FIGUEIREDO et al., 2016), and the subsequent loss of cardioprotection.

Autonomic mechanisms after the RE session are linked to the fall in metabolic demand, such as a gradual withdrawal of sympathetic activity and return of the parasympathetic activity, in order to restore cardiac activity, close to resting levels with

a homeostatic balance (KINGSLEY; FIGUEIROA, 2016). Buchheit et al. (2007) suggested that the contribution of anaerobic metabolism and several other factors during the recruitment of fast-twitch muscle fibers can play a role in autonomic recovery after RE. Some factors may influence such mechanisms as the release of catecholamines, the accumulation of metabolites (lactate, hydrogen ions, inorganic phosphate) and the changes in plasma volume (BUCHHEIT et al., 2007).

In addition, the IPC significantly reduced SNS activity (LFnu) and increased the PSNS activity (HFnu) after the RE session when compared to SHAM and CON protocol at 60-min post. It seems that IPC may accelerate the reactivation of the PSNS after the RE session. In a recent study, Lopes et al. (2018) investigated the effect of IPC on autonomic cardiac recovery after repeated high-intensity sprints. The results showed an increase in the HR recovery in 60 seconds, which shows a higher PSNS activity and a reduction of the SNS activity, which is in agreement with our findings. However, for the variable RMSSDms, which corresponds to the PSNS activity, in the study by Lopes et al. (2018) it was not altered by IPC 6-min post exercise. Incognito et al. (2017) demonstrated that the IPC does not modify the activity of the SNS after isometric strength test in the handgrip, during 3 min of evaluation post exercise. However, the short time taken to evaluate this variable may have interfered in findings of the studies mentioned above, because the PSNS reactivation after exercise session occurs on average 60-min after (KINGSLEY; FIGUEIROA, 2016) and the physiological effects of IPC have a time-dependence correlation (SALVADOR et al., 2016). This correlation can be explained as the reperfusion time after the IPC maneuver is applied, before applying the test or performing any exercise. This correlation is directly proportional such that the longer reperfusion time before starting the test, the greater the ergogenic effect of IPC (SALVADOR et al., 2016). Lisbôa et al. (2017), verified the effects of IPC 1-, 2-, and 8-hour after applying the protocol on the performance of the 50-meter swimming test, the results showed that 2- and 8-hour post exercise generated significantly greater effects when compared to 1 hour after application. This corroborates our study, in which the monitoring of the PSNS activity was done 60 to 120-min after IPC protocol, the recovery was evaluated during 60-min after the RE session, and the IPC has only significantly altered the PSNS activity from 50-min after when compared to the time immediately after the RE session.

In our study, IPC reduced sympathovagal balance at 20-min after exercise when compared to resting value, indicating a higher rate of vagal reactivation. Previous studies

(KINGSLEY; FIGUEIROA, 2016; KINGSLEY et al., 2014; KINGSLEY et al., 2016), that evaluated the sympathovagal balance after an acute RE session, have shown increases when compared to rest, which does not corroborate our findings. In this sense, what may explain the greater speed of the reactivation of the PSNS are the physiological effects generated by the IPC. Enko et al. (2011) showed an increase of the PSNS activity and a reduction of noradrenaline after 30-min of the IPC protocol in the arms. The release of humoral factors in the circulation, such as bradykinin, adenosine, calcitonin, as well as the activation of afferent neural pathways in preconditioned tissues, are the main physiological mechanisms described in the literature (ROSENBERG et al., 2018; BILLAH et al., 2019). Muscle afferent fibers are divided into type III and IV and provide feedback to the cardiovascular system in response to mechanical and metabolic stimulation, respectively (ROSENBERG et al., 2018). Lambert et al. (2016) showed an attenuation of SNS activity after applying the IPC, this activation of neural pathways occurs mainly by the release of adenosine, bradykinin, calcitonin and opioids, the remote preconditioned organ that stimulates the afferent fibers and retransmits the neural signal to the myocardium through the efferent fibers (ROSENBERG et al., 2018; BILLAH et al., 2019).

Neural and humoral mechanisms have been considered an important trigger of PSNS activation induced by IPC (ENKO et al., 2011). Rosenberg et al. (2018) categorize this mechanism into three interrelated events, such as the generation of the cardioprotective signal in the organ or preconditioned tissue, the afferent pathways that transmit this signal to the heart, and the activation of signaling cascades within the heart that mediate the cardioprotective effect. In addition, Gilbey (2007) propose that this mechanism is caused by an "ischemic reflex", which is involved with the activation of sensory receptors of the tissue submitted to ischemia and the increase of the activity of the parasympathetic neural pathways causing cardioprotection.

The present study had also some methodological limitations. The study wasn't recording of muscle sympathetic nerve activity (or the measurement of noradrenaline spillover) is needed to obtain precise information regarding sympathetic tone. Likewise, the use of beta-blockers and muscarinic receptor blockers (like atropine or glycopyrrolate) is required to ascertain if the changes detected through HRV really represent autonomic alterations. Thus, it is recommended that future studies be conducted to elucidate the effects of IPC before a RE session on physiological, hemodynamic and autonomic variables, in order to verify cardiac output, peripheral

vascular resistance, blood pressure, muscle sympathetic nerve activity and heart rate variability, mainly including subjects of different levels of conditioning, age and clinical condition.

This study contributes to the literature on how to prescribe a high intensity RE session for prevention for cardiovascular diseases, through the improvement of the autonomic modulation generated by the IPC maneuver. Therefore, it is recommended to apply 4 cycles of 5-min VO alternated by 4 cycles of 5-min reperfusion before one RE session, consisting of 6 multijoint exercises alternated by segment (3 sets at 80% 1RM until concentric failure). However, the results of this study are likely to apply only to normotensive trained male adults, and further research testing other populations, including hypertensive and heart diseases individuals, is warranted.

4 CONCLUSION

In conclusion, the present results have demonstrated, for the first time, that applying IPC before a high intensity RE session accelerates PSNS recovery in normotensive and trained men. Additionally, because this study used normotensive young subjects, the results presented here may not be generalizable to other populations, such as hypertensive and heart disease subjects. However, it is recommended that future studies be conducted to elucidate the effects of IPC before a RE session on physiological and autonomic variables, in order to verify cardiac output, peripheral vascular resistance and heart rate variability, mainly including subjects of different levels of conditioning, age and clinical condition.

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