ORIGINAL RESEARCH

Yoga Relaxation (*savasana*) decreases cardiac sympathovagal balance in hypertensive patients

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OBJECTIVE: Although relaxation is recommended as complementary therapy for hypertension, its post-intervention cardiovascular autonomic effects are unclear. The objective of this research was to investigate the effects of *savasana* relaxation on cardiovascular autonomic modulation in hypertensive patients.

METHODS: This randomized controlled trial was performed at the Hemodynamic Laboratory of the Physical Education School of the University of São Paulo/Brazil. Sixteen hypertensive (6-women) and 14 normotensive patients (6-women) non-obese subjects participated in 2 random sessions: *savasana* relaxation and control. Patients remained supine for 55 min after interventions. Electrocardiogram, beat-to-beat blood pressure and respiration were acquired during and after interventions for posterior autoregressive spectral analysis of the R-R interval and blood pressure variability.

RESULTS: Hypertensive and normotensive patients presented similar cardiac autonomic modulation responses during and after experimental sessions. During relaxation, low frequency and sympathovagal balance were significantly lower in the Relaxation sessions than during supine rest in the Control sessions. Fifteen minutes after interventions, low frequency and sympathovagal balance were still lower in Relaxation than in Control, and remained lower for 35 min; at 55 min, the variables were similar between sessions. Systolic blood pressure variability did not differ among sessions.

CONCLUSIONS: *Savasana* Relaxation decreases cardiac sympathetic autonomic modulation after its performance in hypertensive patients; this reduction lasts at least 35 minutes and is not blunted in hypertensive patients when compared to normotensive controls. Thus, *savasana* relaxation has positive effects on cardiac autonomic modulation of hypertensive patients, and may be included as a strategy for the non-drug treatment of hypertension.

KEYWORDS: Heart rate variability; Hatha Yoga; Hypertension.

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INTRODUCTION

Hypertension is a highly prevalent disease, afflicting 40% of the American population.[1] It is directly related to acute cardiovascular events, and closely related to end-organ damage.[2] Furthermore, hypertension is related to an autonomic dysfunction, with noted augmented sympathetic and decreased vagal modulation to the heart.[1,3,4]

When regularly performed, relaxation decreases blood pressure[5,6,7,8] sympathetic autonomic modulation to the heart[9] and sympathetic reactivity,[10] being therefore recommended as part of the non-drug treatment of hypertension[1] and for maintaining a good and healthy life style.[11] However, the duration of this acute cardiac autonomic modulation response to relaxation has not been investigated; neither has the possibility that hypertensive patients may be less prone to a decrease in sympathetic and to an increase in parasympathetic heart modulation than normotensive people.

Spectral analysis of heart rate variability has become a useful non-invasive tool to investigate sympathovagal balance to the heart in many circumstances.[12] Studies investigating heart rate variability after relaxation showed a parasympathetic predominance in healthy adults[13] and in post cardiac arrest patients.[14] *Savasana* is an important part of Hatha Yoga relaxation practice. To our knowledge, there is no study addressing the influences of *savasana* on sympathovagal balance of hypertensive subjects. Thus, this aspect still needs

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further investigation, especially in populations which may present cardiac sympathovagal dysfunction.

This paper addresses the following hypothesis: that *savasana* relaxation may decrease cardiac sympathovagal balance in the post-intervention period in hypertensive patients; that this decrease has a measurable duration; and that this decrease of the cardiac sympathetic modulation may be impaired in hypertensive subjects.

Our results show that after *savasana* relaxation, hypertensive patients had a decrease of cardiac sympathovagal balance similar to normotensive controls, and this decrease was present until 35 minutes after intervention.

MATERIALS AND METHODS

Subjects

Sixteen essential stage I hypertensive (HT- 6 women), and 14 normotensive control (NT- 6 women) non-obese patients volunteered. All patients signed a written consent to participate in the study, which was approved by the Ethics Committee of the Heart Institute and by the Ethics Committee of Hospital das Clínicas, Faculty of Medicine, University of São Paulo, Brazil.

Volunteers had their blood pressure measured during two visits to the laboratory. Inclusion criteria were: normotension (accepted if the averages of 3 consecutive measurements were bellow 130 and 85 mmHg for systolic and diastolic blood pressures, respectively) stage I hypertension (averages of 3 consecutive measurements were equal to or higher than 140 and 90 mmHg and below 160 and 100 mmHg), and body mass index (BMI) lower than 30 kg/m.^{2} Hypertensive patients had their medication washed-out 30 days prior to the experiment and replaced by placebo. None of the volunteers were enrolled in any regular exercise program, or were acquainted with the practice of *savasana* relaxation.

In order to establish group pairing related to physical conditioning, peak oxygen uptake (VO_2 peak) was measured directly by a metabolic cart (Medical Graphics Corporation–CAD/NET-2001) during maximal progressive exercise test performed on a cycle ergometer (MIJNHARDT-Ken III) using a protocol of 30 W-increments every 3 min till exhaustion. Heart rate (HR) was continuously monitored by electrocardiography (ECG–TECNOLÓGICA DO BRASIL–SM300). Blood pressure was measured (auscultatory method) at the end of each stage. Tests were interrupted due to physical fatigue, systolic blood pressure exceeding 250 mmHg, diastolic blood pressure greater than 115 mmHg or abnormalities in the electrocardiogram.[15]

Measurements during experimental sessions.

R-R Interval. Electrocardiographic signal was continuously monitored by ECG (TECNOLÓGICA ELETRÔNICA DO BRASIL-SM300).

Blood pressure. Blood pressure was measured by finger photoplethismography on a beat-to-beat basis using a non-invasive finger monitor (OHMEDA-FINAPRES) on the middle finger of the non-dominant arm.

Respiration. Respiratory movements were monitored by a respiratory belt (UFI–Pneumotrace II) positioned around the subject's thorax.

Autonomic modulation. Electrocardiographic, respiratory, and blood pressure were acquired in a microcomputer through the Windaq software - sampling rate of 500 Hz/channel. Sympathetic and parasympathetic cardiac modulations were obtained by spectral analysis of the R-R interval variability, and sympathetic vasomotor modulation was investigated by spectral analysis of systolic blood pressure variability through autoregressive algorithms. On stationary segments of time series, autoregressive parameters were estimated via Levinson-Durbin recursion, and the order of the model chosen according to Akaike's criterion. This procedure permitted us to automatically quantify the center frequency power of each relevant component of the spectrum in absolute as well as normalized units (nu). The normalization procedure was performed by (i) dividing the power of the low-frequency component (LF-0.04-0.15 Hz) or high-frequency component (HF-0.15-0.5 Hz) by the total spectral power; (ii) from this, the power of the very low frequency (VLF–0.00-0.04 Hz) component was subtracted. The result was multiplied by 100.[16]

Intervention.

Relaxation. Relaxation sessions lasted 20 min, based on the *Yoga* technique called *savasana*.[17] During this intervention, volunteers remained supine and tried to develop a conscious detachment from the external environment. To do so, they listened to a recording with 10 min of conducted relaxation followed by 10 min of silence.

Experimental Protocol. All patients were submitted to 2 experimental sessions in a random order, with a minimum 4-day interval. Immediately after arriving at the laboratory, patients emptied their bladders and were prepared for acquisitions. Afterwards, one of the following interventions took place: *savasana* relaxation (R – 20 min) or control (C – 20 min of supine rest). ECG and respiration were recorded continuously for 10 min during R and C. After interventions, patients rested supine during 55 min, which was considered the post-intervention period (P). During this period, ECG, blood pressure, and respiratory signals were continuously recorded during the intervals 15–25 min (P15), 35–45 min (P35), and 50–55 min (P55).

Statistical Analysis. Data were submitted to a two (during interventions) or three-way (after interventions) analysis of variance for repeated measures, establishing groups (normotensive and hypertensive) as independent factors, and experimental sessions (R and C) and stages (post-intervention - P15, P35, P55) as dependent ones. Newman-Keuls test was used for post-hoc comparisons and a level of $P < 0.05$ was accepted as significant. Data are presented as means ± SEM. Results were analyzed with SPSS software v 18.0.

RESULTS

Forty six patients volunteered: 24 hypertensive and 22 normotensive patients. Eight hypertensive patients were excluded: six due to stage II hypertension and systolic and/or diastolic values higher than 220/120 mmHg during rest before the cardiopulmonary exercise test; two due to the use of other medications or presence of atrial fibrillation. Eight normotensive patients were excluded for presenting more than 5 extra-systoles/min (n = 3), or for failing to attend all experimental sessions (n = 5). Thus, 30 patients finished the study, 16 hypertensive (6 women), and 14 normotensive (5 women) ones, as shown in Figure 1.

Physical and cardiovascular characteristics of the patients (normotensive vs. hypertensive patients) are shown in

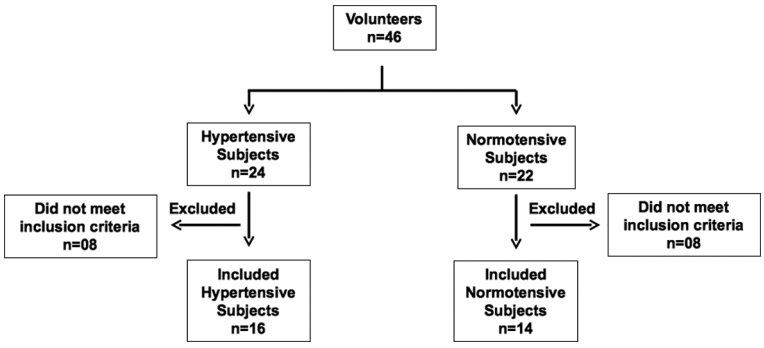

Figure 1 - Subjects distribution. Forty six subjects volunteered: 24 hypertensive, and 22 normotensive subjects. Eight subjects of each group were excluded since they did not meet inclusion criteria; thus, 16 hypertensive, and 14 normotensive subjects were included in this research.

Table 1. Groups were statistically similar for age, body mass index, heart rate, and VO_2 peak. Hypertensive patients had significantly higher systolic and diastolic blood pressures.

Responses during *savasana* relaxation

Heart rate variability and respiration during *savasana* relaxation and control sessions are shown in Table 2. During *savasana*, respiratory frequency was significantly higher in hypertensive vs. normotensive patients. However, in both groups of patients, respiratory frequency during *savasana* was similar to their respective basal levels. Autonomic modulation was similar between normotensive and hypertensive groups during interventions. During Relaxation, total variance was not altered, but LF_{nu} and LF/HF were significantly lower ($p < 0.05$), while RR interval and HF_{nu} were significantly higher than in Control ($p < 0.05$).

Table 1 - Physical and cardiovascular characteristics of normotensive individuals (NT) vs. patients (HT).

	NT	HT
Age, years	39 ± 2	43 ± 1
BMI, kg/m^2	25.3 ± 0.4	26.7 ± 0.6
Heart rate, bpm	74 ± 3	77 ± 2
Systolic Blood Pressure, mmHg	120 ± 1[a]	144 ± 2
Dyastolic Blood Pressure, mmHg	74 ± 1[a]	96 ± 1
VO_2 peak, ml.kg^{-1}.min^{-1}	33.7 ± 1.6	30.5 ± 1.7

Values are means ± SE. BMI – Body mass index. a ≠ HT ($p < 0.05$).

Table 2 - Autonomic data evaluated during *savasana* relaxation (R), and control (C) sessions in the normotensive controls (NT) and hypertensive patients (HT).

	C		R	
	NT	HT	NT	HT
RF	0.26 ± 0.03[b]	0.34 ± 0.07	0.27 ± 0.04[b]	0.32 ± 0.06
RR	922 ± 74	919 ± 34	952 ± 38[a]	913 ± 25[a]
TV_{RR}	2716 ± 715	1910 ± 406	2698 ± 1175	1589 ± 234
LF_{RR}, nu	59 ± 5	67 ± 3	51 ± 5[a]	50 ± 3[a]
HF_{RR}, nu	38 ± 5	29 ± 3	43 ± 5[a]	45 ± 4[a]
LF/HF_{RR}	2.7 ± 0.7	2.7 ± 0.4	1.2 ± 0.3[a]	1.3 ± 0.2[a]

Values are means ± SE. RF – Respiratory Frequency; RR – R-R interval; TV – Total variance; LF – Low frequency; HF – High frequency; LF/HF – Sympathovagal balance. a ≠ C ($p < 0.05$); b ≠ HT ($p < 0.05$).

Responses after *savasana* relaxation

Mean data for the post-intervention period are summarized in Table 3, and the most important variables are shown in Figure 2. Respiratory frequency was significantly lower in the normotensive than in the hypertensive group during all of the post intervention period in all sessions ($p < 0.05$). Respiratory frequencies after interventions were similar between experimental sessions. Hypertensive and normotensive patients presented similar autonomic responses after interventions. Thus, independent of group, mean RR interval was significantly lower after *savasana* versus control at all of the post-intervention periods ($p < 0.05$), while total variance did not change between experimental sessions and measurement points. At P15, LF_{nu} and LF/HF were significantly lower and HF_{nu} was significantly higher after *savasana* vs. control ($p < 0.05$). At P35, LF_{nu} ($p < 0.05$) and LF/HF ($p < 0.05$) were significantly lower and HF_{nu} was significantly higher during *savasana* vs. control ($p < 0.05$). At minute P55 after the interventions, there was no statistical difference in any autonomic variable between experimental sessions. As expected, systolic blood pressure was significantly higher in hypertensive than in normotensive patients ($p < 0.05$). Independent of the post-intervention moment, systolic blood pressure was similar between sessions for *savasana* and control ($p > 0.05$) in both groups. Total variance and LF component of the systolic blood pressure variability were similar among the sessions at all the post-intervention moments.

DISCUSSION

The main findings of the present paper are: 1) *savasana* relaxation caused a reduction of cardiac sympathovagal balance; 2) this shift of the sympathovagal balance toward a higher parasympathetic modulation was similar between hypertensive and normotensive patients; 3) the autonomic cardiac modulation influence of *savasana* relaxation lasted for at least 35 minutes after it was performed; 4) sympathetic vasomotor tone was not influenced by *savasana* relaxation. These results confirm the study hypothesis, since *savasana* relaxation decreased cardiac sympathovagal balance by increasing parasympathetic cardiac modulation and decreasing cardiac sympathetic modulation similarly in hypertensive and normotensive patients. This effect is not impaired in hypertensive patients. Furthermore, the duration of this decrease in sympathetic cardiac modulation

Table 3 - Autonomic data evaluated from 15 to 25 min (P15), from 35 to 45 min (P35), and 50 to 55 min (P55) after the intervention in control (C) and relaxation (R), in the normotensive controls (panel A) and hypertensive (panel B) groups.

A – NT	*Control*			*Savasana Relaxation*		
	P15	*P35*	*P55*	*P15*	*P35*	*P55*
RF, Hz	0.27 ± 0.01[b]	0.28 ± 0.01[b]	0.29 ± 0.01[b]	0.27 ± 0.01[b]	0.28 ± 0.01[b]	0.29 ± 0.01[b]
RR, ms	1038 ± 36	1018 ± 38	1008 ± 38	964 ± 40[a]	950 ± 40[a]	945 ± 36[a]
TV_{RR}, ms^2	4009 ± 948	3160 ± 712	3929 ± 773	3876 ± 1839	3814 ± 1219	2955 ± 1024
LF_{RR}, nu	61 ± 4	66 ± 3	57 ± 6	50 ± 6[ad]	52 ± 5[a]	55 ± 6
HF_{RR}, nu	31 ± 4	31 ± 4	38 ± 5	41 ± 5[ad]	41 ± 6[a]	40 ± 6
LF/HF_{RR}	3.0 ± 0.8	2.8 ± 0.6	2.4 ± 0.5	2.2 ± 0.6[ad]	2.3 ± 0.7[a]	2.2 ± 0.5
SBP, mmHg	111 ± 5[b]	117 ± 5[b]	116 ± 5[b]	109 ± 3[b]	111 ± 4[b]	116 ± 4[b]
TV_{SBP}, $mmHg^2$	31 ± 6	24 ± 3	25 ± 4	30 ± 4	42 ± 10	35 ± 8
LF_{SBP}, $mmHg^2$	19 ± 7	13 ± 3	18 ± 5	12 ± 3	17 ± 10	26 ± 8
B – HT	*Control*			*Savasana Relaxation*		
	P15	*P35*	*P55*	*P15*	*P35*	*P55*
RF, Hz	0.35 ± 0.01	0.33 ± 0.01	0.33 ± 0.01	0.32 ± 0.01	0.32 ± 0.02	0.32 ± 0.01
RR, ms	948 ± 29	944 ± 32	968 ± 32	937 ± 28[a]	934 ± 26[a]	926 ± 24[a]
TV_{RR}, ms^2	2793 ± 519	3026 ± 618	2746 ± 611	2960 ± 937	2719 ± 607	2110 ± 543
LF_{RR}, nu	68 ± 3	68 ± 2	58 ± 4	46 ± 4[a]	48 ± 3[a]	54 ± 5
HF_{RR}, nu	27 ± 3	27 ± 2	35 ± 4	46 ± 4[a]	45 ± 3[a]	40 ± 5
LF/HF_{RR}	3.3 ± 0.5	2.8 ± 0.3	2.4 ± 0.5	1.2 ± 0.2[a]	1.3 ± 0.2[a]	2.3 ± 0.5
SBP, mmHg	130 ± 4	132 ± 4	138 ± 4	134 ± 4	130 ± 4	138 ± 5
TV_{SBP}, $mmHg^2$	35 ± 5	42 ± 1	24 ± 7	44 ± 8	35 ± 6	28 ± 5
LF_{SBP}, $mmHg^2$	19 ± 4	10 ± 2	11 ± 3	24 ± 7	15 ± 6	12 ± 4

Values are means ± SE. RF – respiratory frequency (Hz); RR – R-R interval (msec); TV – total variance ($msec^2$); LF – low frequency component; HF – high frequency component; LF/HF – sympathovagal balance; SBP – systolic blood pressure (mmHg). a ≠ C ($p < 0.05$); b ≠ HT ($p < 0.05$).

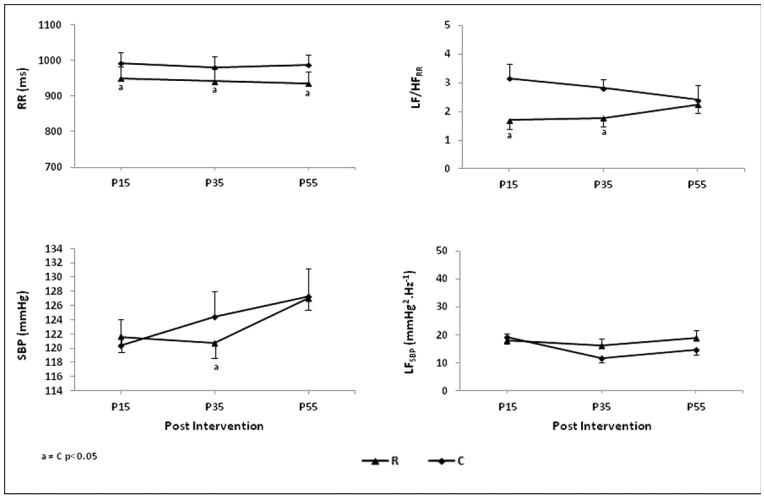

Figure 2 - RR interval, sympathovagal balance of RR interval variability (LF/HF_{RR}), systolic blood pressure (SBP) and low frequency component of systolic blood pressure (LF_{SBP}) measured at 15 (P15), 35 (P35), and 55 (P55) minutes of post-intervention period on the two experimental sessions: relaxation (R) and control (C). Data from both groups (normotensive and hypertensive patients) were combined.

could be measured. It lasted at least 35 minutes. Thus, it is possible to state that *savasana* relaxation may have clinical importance for the non-drug treatment of hypertension.

In accordance with the literature,[5,6] relaxation led to a decrease in LF_{nu} and to an increase in HF_{nu} during its execution, indicating a shift of the sympathovagal balance towards the parasympathetic branch of cardiac autonomic modulation. Furthermore, *savasana* relaxation also led to a decrease in sympathetic and to an increase in parasympathetic cardiac modulation after its execution, because 35 min after relaxation, HF_{nu} was higher and LF_{nu} and LF/HF were lower than in C. Because there was no difference for autonomic modulation between groups either during, or after intervention, the hypothesis that hypertensive patients might have impaired autonomic response to relaxation is refuted. During relaxation, LF/HF was already below C and remained low until 35 min of the post-intervention period. However, this status progressively changed throughout this period, since sympathovagal balance returned to control levels at 55 min of post-intervention. Thus, it is possible to state that the duration of the effect is at least 35 minutes.

The mechanisms by which *savasana* relaxation may have decreased sympathovagal balance after its execution is out of the scope of this study. However, some speculation may be

pertinent. One could state that the decrease in sympathovagal modulation during relaxation and after intervention might be related to changes in respiratory frequency, which markedly influences cardiac autonomic modulation.[18] However, as respiratory frequency was not altered by relaxation, and was similar after intervention, this possibility is refuted. The effect of relaxation, thus, could be related to its effects on the central regulator of sympathetic and parasympathetic nucleus.[19] Nevertheless, further studies are necessary to investigate this issue.

Another possible mechanism for cardiovascular changes after intervention might have been altered vasomotor tone, as is observed after physical exercise.[10,20] In fact, relaxation led to hypotension, as systolic blood pressure was lower after Relaxation than after Control. Previous studies,[21,22] employing microneurography suggested that the post-exercise blood pressure decrease is due to a decrease in peripheral sympathetic activity when recovery is conducted in a supine position. However, in the present study, total variance and LF component of systolic blood pressure variability, which may represent sympathetic vasomotor activity,[12] were not decreased after relaxation. These different results may be related to methodological aspects: 1) the kind of measurement, i.e. microneurography measures sympathetic traffic to a specific nerve, while blood pressure variability evaluates vasomotor response to sympathetic activity in the whole body, and 2) the timing, i.e. previous studies measured sympathetic activity 60 min after exercise, while in the present study measurements were taken earlier, and did not address exercise conditions. Thus, it seems pertinent to state that mechanisms responsible for cardiovascular autonomic modulation effects of *savasana* relaxation are different from those of physical exercise.

Since autonomic dysfunction is associated with a worse cardiovascular prognosis, and hypertensive patients may have increased cardiac sympathovagal balance,[3,4] the results of this study may have clinical importance for hypertensive patients. The investigated intervention (*savasana* relaxation) decreased sympathovagal balance after its execution, and this reduction was not impaired by hypertension. Thus, these findings strengthen the recommendation of relaxation as part of the non-pharmacological treatment of hypertension, performed alone or in association.

CONCLUSION

In conclusion, *savasana* relaxation decreases cardiac sympathovagal balance after its execution in hypertensive patients. This reduction lasts at least 35 minutes, and is not impaired in hypertensive when compared to normotensives patients. Thus, *savasana* relaxation has positive effects on cardiac autonomic modulation of hypertensive patients, and may be included as a strategy for the non-drug treatment of hypertension.

Limitations

Both men and women were studied and, thus, differences between genders were not addressed. Results are limited to stage I hypertensive and healthy normotensive patients.

Variables were not measured before interventions, which would have allowed a specific description of what happened from pre to post-intervention periods. However, as sessions were randomly ordered, it is reasonable to expect that pre-intervention values were similar and thus comparisons between sessions at the post-intervention period reflect differences in behaviors between pre and post periods.

Spectral analysis of cardiovascular variability must always be faced as a tool to access cardiovascular autonomic modulation and not total autonomic activity.

The different groups were expected to present different cardiac autonomic modulations at rest, with a greater sympathetic modulation in hypertensive patients,[3,4] but this was not corroborated by our results. One possible reason for this is the hypertension stage of the sample: because patients had to be washed out of medication 1 month prior to the protocol, only stage I hypertensive patients without complications entered the study. Most of the literature addresses stage II hypertensive patients.

SUMMARY

Briefly, *savasana* relaxation decreases cardiac sympathovagal balance (LF/HF ratio), increasing parasympathetic modulation to the heart in hypertensive patients, without disadvantages when compared to normotensives. This effect lasts at least 35 minutes. Its mechanisms require further investigation.

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AUTHOR DISCLOSURE STATEMENT

No competing financial interests exist.

RESUMO

OBJETIVO: Embora o relaxamento seja recomendado como terapia complementar no tratamento da hipertensão, seus efeitos na modulação autonômica cardiovascular ainda não estão claros. O objetivo desse trabalho foi investigar os efeitos do relaxamento na modulação autonômica cardíaca de indivíduos hipertensos.

MÉTODOS: Este estudo aleatorizado e controlado foi realizado no Laboratório de Hemodinâmica da Escola de Educação Física e Esporte da Universidade de São Paulo/Brasil. Dezesseis hipertensos (6-mulheres) e 14 normotensos controle (6-mulheres) não-obesos participaram de 2 sessões aleatórias: relaxamento (R) e controle (C). Os participantes permaneceram supinos por 55 minutos após as intervenções. Eletrocardiograma, pressão arterial batimento a batimento, e respiração foram adquiridos durante e após as intervenções para a posterior análise espectral autorregressiva das variabilidades do intervalo R-R e da pressão arterial.

RESULTADOS: Hipertensos e normotensos apresentaram respostas semelhantes de modulação autonômica cardíaca durante e após as sessões experimentais. Durante o relaxamento, a baixa frequência e o equilíbrio simpatovagal da variabilidade da frequência cardíaca estavam significantemente mais baixas no R do que no repouso supino em C. Quinze minutos após a intervenção a baixa frequência e o equilíbrio simpatovagal ainda estavam inferiores em R do que em C, o que ocorreu também nos 35 minutos, mas não aos 55 minutos. A variabilidade da pressão arterial foi semelhante entre as sessões.

CONCLUSÃO: O relaxamento *savasana* reduz a modulação autonômica simpática cardíaca de hipertensos; essa redução perdura por pelo menos 35 minutos após o término do relaxamento e não é prejudicada nos hipertensos quando comparados com normotensos. Dessa forma, o relaxamento *savasana* tem efeitos positivos na modulação autonômica cardíaca de hipertensos e pode ser incluído como estratégia para o tratamento não-medicamentoso da hipertensão arterial.

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