

Effects of exercise intensity and creatine loading on post-resistance exercise hypotension

Efeitos da intensidade do exercício e da sobrecarga de creatina na hipotensão pós-exercício resistido

Juliano Rodrigues Moreno^{1,2}
 Gisela Arsa da Cunha^{1,2}
 Pedro Luiz Braga²
 Juliana Hott de Fúcio Lizardo³
 Carmen Silvia Grubert Campbell¹
 Mara Lucy Dompietro Ruiz Denadai²
 Herbert Gustavo Simões¹

Resumo – A hipotensão pós-exercício tem um importante papel no tratamento não-farmacológico da hipertensão. Ela é caracterizada por redução na pressão arterial após uma única sessão de exercício em relação aos valores basais. Esse estudo analisou os efeitos da intensidade do exercício e da suplementação de creatina na hipotensão pós-exercício, assim como, o provável papel do lactato sanguíneo nessa resposta. Participaram do estudo dez indivíduos normotensos que realizaram sessões de exercício resistido (ER) antes (AC) e após (PC) suplementação de creatina: 1. Resistência muscular (RM) – 30 repetições a 30% de 1 repetição máxima; 2. Hipertrofia (HP) – 8 repetições a 75% de 1 repetição máxima. A pressão arterial foi mensurada antes e após o exercício. O lactato sanguíneo foi mensurado no período pós-exercício. As sessões de RM e HP promoveram diminuição da pressão arterial sistólica ($\Delta -19 \pm 1,0$ mmHg; $\Delta -15 \pm 0,9$ mmHg, respectivamente, $P < 0,05$) a qual foi atenuada após suplementação de creatina ($\Delta -7,1 \pm 1,0$ mmHg; $\Delta -11 \pm 1,0$ mmHg, respectivamente, $P < 0,05$). O pico de lactato foi atenuado após suplementação de creatina na sessão HP (AC $19 \pm 0,4$ mM; PC $15 \pm 0,4$ mM, $P < 0,05$) e permaneceu inalterado após suplementação de creatina na sessão RM (AC $16 \pm 0,8$ mM; PC $14 \pm 0,4$ mM, $P > 0,05$). Concluiu-se que a intensidade do exercício resistido não influenciou a hipotensão pós-exercício. A suplementação de creatina atenuou a redução dos valores pressóricos pós-exercício resistido. Além disso, os resultados sugerem a participação do lactato sanguíneo na hipotensão pós-exercício resistido.

Palavras-chave: Pressão arterial; Exercício; Ácido láctico; Creatina.

Abstract – Postexercise hypotension plays an important role in the non-pharmacological treatment of hypertension and is characterized by a decrease in blood pressure after a single exercise bout in relation to pre-exercise levels. This study investigated the effects of exercise intensity and creatine monohydrate supplementation on postexercise hypotension, as well as the possible role of blood lactate in this response. Ten normotensive subjects underwent resistance exercise sessions before (BC) and after (AC) creatine supplementation: 1) muscle endurance (ME) consisting of 30 repetitions at 30% of one-repetition maximum; 2) hypertrophy (HP) consisting of 8 repetitions at 75% of one-repetition maximum. Blood pressure was measured before and after the exercise bout. Blood lactate was measured after the exercise bout. The HP and ME sessions promoted a decrease in systolic blood pressure ($\Delta -19 \pm 1.0$ mmHg; $\Delta -15 \pm 0.9$ mmHg, respectively, $P < 0.05$), which was attenuated after creatine supplementation ($\Delta -7.1 \pm 1.0$ mmHg; $\Delta -11 \pm 1.0$ mmHg, respectively, $P < 0.05$). Peak blood lactate was attenuated after creatine supplementation in the HP session (BC: 19 ± 0.4 mM; AC: 15 ± 0.4 mM, $P < 0.05$) and remained unchanged after creatine supplementation in the ME session (BC: 16 ± 0.8 mM; AC: 14 ± 0.4 mM, $P > 0.05$). In conclusion, resistance exercise intensity did not influence postexercise hypotension. Creatine supplementation attenuated the decrease in blood pressure after resistance exercise. The results suggest the involvement of blood lactate in post-resistance exercise hypotension.

Key words: Blood pressure; Exercise; Lactic Acid; Creatine.

1 Laboratory of Physical Education, Department of Physical Education, Catholic University of Brasília, Brasília, DF, Brazil.

2 Laboratory of Exercise Physiology, Department of Physical Education, University of Mogi das Cruzes, Mogi das Cruzes, SP, Brazil.

3 Laboratory of Cardiac Electromechanics, Department of Physiological Sciences, Federal University of Espírito Santo, Vitória, ES, Brazil.

Recebido em 18/12/07
 Aprovado em 24/07/08

INTRODUCTION

Recently, attention has been focused not only on the cardiovascular benefits of physical training, but also on the effects of one acute exercise session. After an acute exercise bout, blood pressure (BP) levels are reduced for minutes or hours in relation to pre-exercise levels^{1,2}. This phenomenon is called postexercise hypotension (PEH) and has been widely investigated because of its importance for the treatment and prevention of arterial hypertension^{3,4}.

The possible mechanisms underlying PEH include a reduction of sympathetic nerve activity and decreased vascular responsiveness to α -adrenergic receptor activation, which elicit a sustained reduction of peripheral vascular resistance⁵. Taking into account vasorelaxation effects, local substances released by exercising muscles may also be involved in PEH. Hussain et al.⁶ investigated the effects of circulating metabolites such as blood lactate on alterations in femoral artery blood flow and vascular tone during recovery from high-intensity exercise (Wingate test). The authors demonstrated that blood lactate levels remained elevated during the period of persistent vasodilatation after the exercise bout.

Supplementation with creatine monohydrate can increase intramuscular phosphocreatine stores by about 25%, thus optimizing the resynthesis of adenosine triphosphate by the anaerobic alactic system⁷. In addition, creatine supplementation results in intramuscular water retention, causing hypertrophy, and in increased protein synthesis, improving contractile capacity and performance during short-term high intensity exercise^{7,8}. Creatine supplementation has also been associated with lower blood lactate accumulation during high-intensity exercise⁹. However, the effects of blood lactate and creatine supplementation on the BP response after a single bout of resistance exercise are unclear. Moreover, the influence of resistance exercise intensity on PEH is controversial^{10, 11}.

Therefore, the objective of the present study was to investigate the effects of exercise intensity and creatine supplementation on PEH. Since creatine supplementation may decrease blood lactate accumulation, we also measured this metabolite after exercise and evaluated the putative role of blood lactate in PEH.

METHODOLOGICAL PROCEDURES

Subjects

Ten healthy men (23.0 ± 1.9 years, 175.3 ± 5.3 cm,

78.4 ± 6.3 kg) volunteered to participate in the study. All subjects received detailed information about the procedures, risks and benefits of the study, and signed an informed consent form previously approved by the Local Ethics Committee for Human Research. All participants were experienced body builders.

Exercise protocols

All participants underwent four exercise sessions as follows: 1) hypertrophy session before supplementation with creatine monohydrate (HP BC); 2) hypertrophy session after creatine supplementation (HP AC); 3) endurance session before creatine supplementation (ME BC); 4) endurance session after creatine supplementation (ME AC). The hypertrophy session consisted of 3 sets of 8 repetitions at 75% of one-repetition maximum (1RM), whereas the endurance session consisted of 3 sets of 30 repetitions at 30% of 1RM. The following types of exercise were performed: straight supine, leg press, stand-up-rowing in the low, knee extension, knee curl exercise, and elbow curl. The exercise sessions were performed randomly at intervals of 48 h between sessions.

1RM test

The 1RM test was performed in the following sequence for all exercises: straight supine, leg press, stand-up-rowing in the low, knee extension, knee curl exercise and elbow curl. Three trials were performed for each exercise at intervals of 3-5 minutes. An initial load (kg) lower than that usually used for training was determined for the 1RM test. Next, two sets of exercise were performed and the load was progressively increased in subsequent sets. The subject was allowed to rest for 3-5 minutes between sets. The 1RM was defined as the maximum weight that could be lifted only once.

Creatine supplementation

The subjects ingested 5 g creatine mixed with 40 g sucrose diluted in 300 ml water, 4 times a day for 5 days (loading phase). Previous results from our laboratory demonstrated that this supplementation evoked a significant increase in muscle mass.

Blood pressure and blood lactate measurement

Systolic blood pressure (SBP) and diastolic blood pressure (DBP) were measured by the indirect auscultatory method using a sphygmomanometer and stethoscope. BP was assessed at 5-minute intervals for 20 minutes before the exercise bout

and at 15-minute intervals for 90 minutes after the exercise bout. Baseline BP was calculated as the average of pre-exercise measurements. During the measurements, the volunteers remained seated on a comfortable couch in an environment without noise or variations in temperature. All measurements were performed by the same researcher.

For the determination of blood lactate concentration, 25 μ l capillary blood was drawn from the ear lobe after aseptis and transferred to an Eppendorf tube containing 50 μ l 1% NaF. Blood samples were collected before the beginning of the sessions, at 3-minute intervals during the first 15 minutes of recovery, and 30, 45, 60, 75 and 90 minutes after exercise. Blood lactate was measured by an electric enzymatic method.

Statistical analysis

The results are reported as means \pm standard error of the mean (SEM). BP was compared before and after exercise by two-way ANOVA for repeated measures. Absolute changes in BP from pre-exercise levels

and peak blood lactate were analyzed by two-way randomized ANOVA. When significant differences were detected, post hoc analyses were performed using Tukey's least significant difference test. A P value < 0.05 was considered to be significant.

RESULTS

Postexercise blood pressure responses

Figure 1 shows the mean SBP and DBP values before and after the exercise bout obtained for the HP BC, HP AC, ME BC and ME AC sessions. A decrease in SBP was observed after the HP BC session throughout post-exercise bout ($P < 0.05$), which was abolished after creatine supplementation. A similar response was observed after the ME sessions, i.e., creatine supplementation abolished hypotension. After the HP BC and ME BC sessions, DBP decreased during the first 30 minutes in post-exercise bout. The changes in SBP from baseline (pre-exercise) to 90-minute postexercise were $\Delta -19 \pm 1.0$ mmHg ($P < 0.05$) for HP BC, Δ

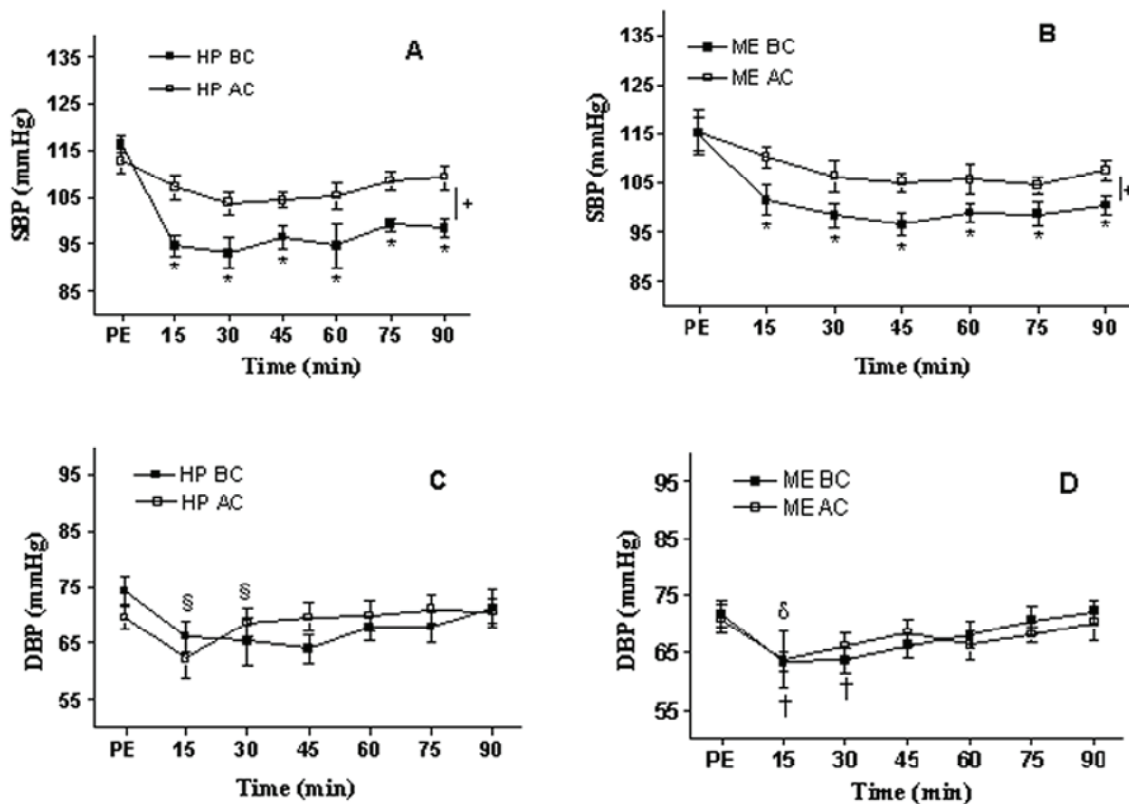


Figure 1. A: Systolic blood pressure (SBP) before (PE) and at 15-minute intervals after the resistance exercise bout for hypertrophy sessions before (HP BC) and after (HP AC) creatine supplementation. B: SBP before (PE) and at 15-minute intervals after the exercise bout for endurance sessions before (ME BC) and after (ME AC) creatine supplementation. C: DBP before (PE) and at 15-minute intervals after the exercise bout for HP BC and HP AC sessions. D: DBP before (PE) and at 15-minute intervals after the exercise bout for ME BC and ME AC sessions. Data are reported as means \pm SEM. * $P < 0.05$ vs PE; + $P < 0.05$ HP BC vs HP AC and ME BC vs ME AC. $\S P < 0.05$ vs PE for HP BC; $\delta P < 0.05$ vs PE for ME AC; $\ddagger P < 0.05$ vs PE for ME BC.

-7.1 ± 1.0 mmHg ($P < 0.05$) for HP AC, $\Delta -16 \pm 0.9$ mmHg ($P < 0.8$) for ME BC, and $\Delta -9.3 \pm 0.9$ mmHg ($P < 0.05$) for ME AC (Figure 2).

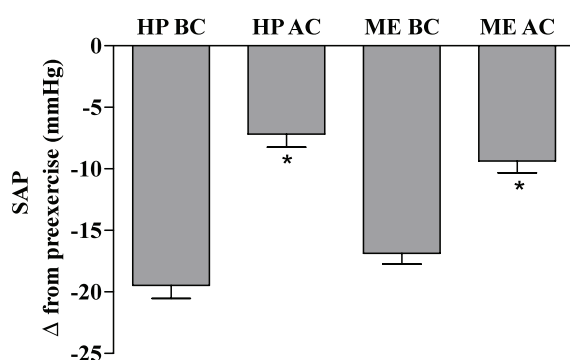


Figure 2. Mean absolute change (Δ) in systolic blood pressure (SBP) from pre-exercise (20 minutes) to 90-minute postexercise bout obtained for sessions of hypertrophy before (HP BC) and after (HP AC) creatine supplementation and sessions of endurance before (ME BC) and after (ME AC) creatine supplementation. Data are reported as means \pm SEM. * $P < 0.05$ vs before creatine supplementation.

Blood lactate

Creatine supplementation attenuated peak blood lactate after the HP session ($P < 0.05$). No change in blood lactate concentration was observed after creatine supplementation for the ME session ($P > 0.05$).

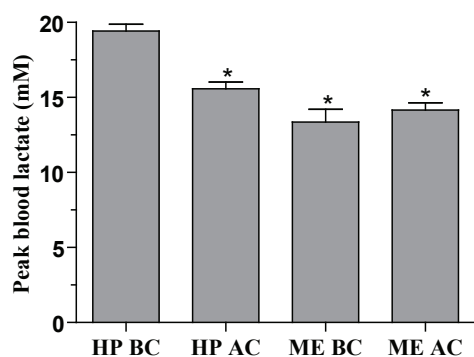


Figure 3. Peak blood lactate after sessions of hypertrophy before (HP BC) and after (HP AC) creatine supplementation and sessions of endurance before (ME BC) and after (ME AC) creatine supplementation. Data are reported as means \pm SEM. * $P < 0.05$ vs HP BC.

DISCUSSION

This study investigated the effects of resistance exercise of high (HP) and low (ME) intensity associated or not with creatine supplementation on PEH. The HP and ME sessions elicited a similar PEH response, but creatine supplementation attenuated the hypotensive effect of resistance exercise. We also evaluated the influence of blood lactate on PEH and the results obtained suggest a possible role of this metabolite in this response.

Despite controversies regarding the occurrence of a decrease in resting BP after acute resistance exercise^{13,14} the hypotensive response observed in the present study agrees with some results reported for humans and animals¹⁵⁻²⁰. The effect of exercise intensity on PEH is also controversial^{10,11}. Most studies investigating the effects of exercise intensity have used aerobic exercise, and reports evaluating the influence of this parameter on the BP decrease after acute resistance exercise are scarce. In the present study, exercise performed at different intensities caused a similar decrease in BP during recovery. This finding agrees with Brown et al.¹⁶ who observed no differences in PEH after resistance exercise performed at 40% and 60% of 1RM.

Creatine supplementation is an ergogenic aid widely used to boost exercise performance by improving the tension capacity of skeletal muscle, resulting in lower metabolic stress, i.e., lower local metabolite production²¹. It has been well established that local metabolites and substances released in the vascular bed of exercising muscles mediate important vasodilatation during and after an exercise bout. Our results showed that creatine supplementation abolished PEH in both exercise sessions. Based on this finding, our hypothesis is that local metabolite release might be reduced after creatine supplementation, with consequent lower vascular relaxation and abolishment of PEH. Supporting this mechanism, the present results also demonstrated that creatine supplementation attenuated peak blood lactate after the HP session. These findings are in agreement with previous studies^{9,2} showing that creatine supplementation is associated with lower blood lactate accumulation during high-intensity exercise. Moreover, Hussain et al.⁶ demonstrated that an increase in blood lactate after exercise was associated with a decrease in femoral artery vascular resistance. Lactic acid dissociates to H^+ ions and these ions may activate ATP-sensitive K^+ (K_{ATP}) channels present in vascular smooth muscle. The activation of these channels may lead to smooth muscle hyperpolarization and, consequently, to vascular relaxation and a decrease in BP. K_{ATP} channels may also be activated by other endogenous vasodilators such as prostacyclin, adenosine and nitric oxide²². Since a good part of the blood flow is deviated to skeletal muscle during and postexercise, metabolite-induced vascular resistance decreases at this site and might affect systemic BP. Thus, our results suggest that PEH was abolished after creatine supplementation because blood lactate was reduced during the HP

session. However, although the BP decrease observed after the ME session was abolished after creatine supplementation, peak blood lactate remained unchanged. The mechanism involved in the BP decrease after HP sessions seems to be an increase in blood lactate; however, this mechanism does not explain the hypotension observed after exercise sessions of lower intensity. Since high-intensity exercise elicits higher blood lactate accumulation than exercise of lower intensity, different factors might be responsible for the decrease in BP. In view of the increase in blood volume and, consequently, in cardiac output, the water retention caused by creatine supplementation^{7,8} may also be related to the attenuation of PEH. However, Powers et al.²³ observed that, although creatine supplementation caused significant body water retention, it did not affect body fluid distribution. Other studies found no change in resting BP after creatine supplementation when compared to a similar situation without supplementation^{24,25}. Since creatine supplementation decreases the release of several local metabolites, other mediators may be involved in PEH during the ME session. Nitric oxide is an important vasodilator that seems to exert an influence on PEH as demonstrated in a previous investigation²⁶. Further studies are necessary to determine which metabolites are influenced by creatine supplementation.

CONCLUSION

The present results demonstrated an important decrease in resting BP after acute resistance exercise. This response was not influenced by exercise intensity, but was abolished after creatine supplementation. Since peak blood lactate was attenuated after creatine supplementation in the high-intensity session, this metabolite might be involved in PEH. Thus, this study suggests that creatine supplementation abolishes the beneficial effects of acute resistance exercise on BP.

REFERENCES

1. Kenney MJ, Seals DR. Postexercise hypotension: key features, mechanisms and clinical significance. *Hypertension* 1993;22(5):653-664.
2. MacDonald JR. Potential causes, mechanisms and implications of post exercise hypotension. *J Hum Hypertens* 2002;16(4):225-236.
3. Halliwill JR. Mechanisms and clinical implications of post-exercise hypotension in humans. *Exerc Sports Sci Rev* 2001;29(2):65-70.
4. Pescatello LS, Franklin BA, Fagard RH, Farquhar WB, Kelley GA, Ray CA. Exercise and hypertension: Position stand of American College of Sports Medicine. *Med Sci Sports Exerc* 2004;36(3):533-553.
5. Halliwill JR, Taylor JA, Eckberg DL. Impaired sympathetic vascular regulation in humans after acute dynamic exercise. *J Physiol* 1996;495(Pt 1):279-288.
6. Hussain ST, Smith RE, Medbak S, Wood RF, Whipp BJ. Haemodynamic and metabolic responses of the lower limb after high intensity exercise in humans. *Exp Physiol* 1996;81(2):173-187.
7. Greenhaff PL. Creatine and its application as an ergogenic aid. *Int J Sport Nutr* 1995;5(5):S100-110.
8. Willoughby DS, Rosene JM. Effects of oral creatine and resistance training on myogenic regulatory factor expression. *Med Sci Sports Exerc* 2003;35(6):923-929.
9. Balsom PD, Soderlund K, Sjodin B, Ekblom B. Skeletal muscle metabolism during short duration high-intensity exercise: influence of creatine supplementation. *Acta Physiol Scand* 1995;154(3):303-310.
10. Pescatello LS, Guidry MA, Blanchard BE, Farquhar WB, Kelley GA, Ray CA. Exercise intensity alters postexercise hypotension. *J Hypertens* 2004;22(10):1881-1888.
11. Forjaz CL, Matsudaira Y, Rodrigues FB, Nunes N, Negrão CE. Post-exercise changes in blood pressure, heart rate and rate pressure product at different exercise intensities in normotensive humans. *Braz J Med Biol Res* 1998;31(10):1247-1255.
12. Focht BC, Koltyn KF. Influence of resistance exercise of different intensities on state anxiety and blood pressure. *Med Sci Sports Exerc* 1999;31(3):456-463.
13. Roltsh 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;33(6):881-886.
14. Melo CM, Alencar Filho AC, Tinucci T, Mion DJr, Forjaz CL. Postexercise hypotension induced by low-intensity resistance exercise in hypertensive women receiving captopril. *Blood Press Monit* 2006;11(4):183-189.
15. Brown SP, Clemons JM, He Q, Liu S. Effects of resistance exercise and cycling on recovery blood pressure. *J Sports Sci* 1994;12(5):463-468.
16. 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.
17. MacDonald JR, MacDougall JD, Interisano SA, Smith KM, McCartney N, Moroz JS. Hypotension following mild bouts of resistance exercise and submaximal dynamic exercise. *Eur J Appl Physiol* 1999;79(2):148-154.
18. Fisher MM. The effect of resistance exercise on recovery blood pressure in normotensive and borderline hypertensive women. *J Strength Cond Res* 2001;15(2):210-216.
19. Stout J, Eckerson J, Ebersole K. Effect of creatine loading on neuromuscular fatigue threshold. *J Appl Physiol* 2000;88(1):109-112.
20. Ceddia RB, Sweeney G. Creatine supplementation increases glucose oxidation and AMPK phosphorylation and reduces lactate production in L6 rat skeletal muscle cells. *J Physiol* 2004;555(Pt 2):409-421.

21. Quayle JM, Standen NB. K⁺ATP channels in vascular smooth muscle. *Cardiovasc Res* 1994;28(6):797-804.
22. Powers ME, Arnold BL, Weltman AL, Perrin DH, Mistry D, Kahler DM, et al. Creatine supplementation increases total body water without altering fluid distribution. *J Athl Train* 2003;38(1):44-50.
23. Mihic S, Macdonald Jr, McKenzie S, Tarnopolsky MA. Acute creatine loading increases fat-free mass, but does not affect blood pressure, plasma creatinine, or CK activity in men and women. *Med Sci Sports Exerc* 2000;32(2):291-296.
24. Peeters BM, Lantz CD, Mayhew JL. Effect of oral creatine monohydrate and creatine phosphate supplementation on maximal strength indices, body composition, and blood pressure. *J Str Cond Res* 1999;13(1):3-9.
25. Rao SP, Collins HL, DiCarlo SE. Postexercise α -adrenergic receptor hyporesponsiveness in hypertensive rats is due to nitric oxide. *Am J Physiol Integrative Comp Physiol* 2002;282(4):R960-R968.

Endereço para correspondência

Juliana Hott de Fúcio Lizardo
Universidade Federal do Espírito Santo, Departamento de Ciências Fisiológicas Laboratório de Eletromecânica Cardíaca
Av. Marechal Campos, 1468
Maruípe
29042-751 – Vitória, Espírito Santo, Brasil.
E-mail: julianahott@yahoo.com