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ORIGINAL ARTICLE

Hemodynamic effects of short-term arm-cranking exercise training in hypertensive type-2 diabetics

Effets hémodynamiques d'un exercice sur ergomètre à bras chez des diabétiques hypertendus

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KEYWORDS

Type-2 diabetes;
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Upper limb exercise

Summary

Objectives. – To investigate resting heart rate, systolic and diastolic blood pressure (SBP and DBP), pulse pressure (PP), and rate-pressure product (RPP), as well as the occurrence of post-exercise hypotension (PEH) before and after 4 weeks of arm-cranking exercise training in hypertensive type-2 diabetics.

Methods. – Sixteen individuals were allocated in trained (TRA; $n=10$) or control group (CON; $n=6$). They performed two submaximal incremental tests before (IT-1) and after (IT-2) 4 weeks of arm-cranking exercise training or non-training. BP was measured at rest and for 1 h after each IT.

Results. – Both groups presented PEH of SBP, DBP and MAP after IT-1 and this response was attenuated after IT-2 for group TRA, but not for CON. There were reductions in resting BP, RPP and PP after the 4-week intervention for group TRA, with no changes for CON.

Conclusion. – Hypertensive type-2 diabetics presented PEH after a single bout of arm-cranking exercise, but the effect was attenuated after 4 weeks of training. This might be a result of the significant reduction in resting BP, PP and RPP observed on IT-2 for group TRA. These results suggest that arm-cranking exercise may be effective to promote both acute and chronic hemodynamic benefits for this population.

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MOTS CLÉS

Diabétique de type 2 ;
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Hypotension
post-exercice ;
Exercice de membres
supérieurs

Résumé

Objectifs. – Il s'agit d'examiner, chez des diabétiques de type 2 hypertendus, la fréquence cardiaque, la pression artérielle systolique et diastolique (SBP et DBP), la pression pulsée (PP) et le double produit (RPP) au repos ainsi que l'hypotension post-exercice (PEH), avant et après sur un exercice de pédalage des membres supérieurs sur ergomètre adapté.

Méthodes. – Seize sujets ont été assignés à un groupe de sujets entraînés (TRA, $n=10$) ou témoins (CON, $n=6$). Ils ont effectué deux exercices incrémentaux sous-maximaux avant (IT-1) et après (IT-2) quatre semaines d'entraînement de pédalage de bras (ou pas pour le groupe CON). Les SBP et DBP ont été mesurées au repos et une heure après chaque IT.

Résultats. – Les sujets des deux groupes ont présenté une PEH de la SBP, DBP et pression artérielle moyenne (PAM) après IT-1. Une réponse atténuée de ces variables hémodynamiques est observée uniquement dans le groupe TRA après IT-2. On observe de même, une réduction de SBP, DBP, RPP et PP mesurés au repos après les quatre semaines d'entraînement, sans aucune modification dans le groupe CON.

Conclusion. – Les diabétiques de type 2 hypertendus présentent une hypotension artérielle après un exercice sur un ergomètre de membres supérieurs (PEH), qui est atténuée après quatre semaines d'entraînement d'un tel exercice. Cela peut être dû à la réduction de la BP, PP et/ou du RPP de repos. Ces résultats suggèrent que l'exercice sur ergomètre pour bras est efficace afin de corriger les altérations hémodynamiques observées chez ces patients.

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1. Introduction

Type-2 diabetes (T2D) is a high prevalent disease often associated to arterial hypertension (AH). Chronic hyperglycemia may lead to autonomic and endothelial dysfunction, atherosclerosis and increased inflammation, sodium retention and intracellular calcium levels in the smooth and cardiac muscles [1,2], contributing to augments in blood pressure (BP).

Concomitant T2D and AH render individuals more prone to end-organ injuries and cardiovascular events, decreasing life quality and expectancy [3]. Therefore, besides blood glucose, an effective BP control is also mandatory for these individuals to avoid or attenuate secondary complications. To help achieving this goal, physical exercise is an effective non-pharmacological strategy, with both acute [4] and chronic [5,6] effects on BP.

The acute reduction of BP after a single exercise bout, called post-exercise hypotension (PEH) [7], may be sustained for as long as 24 h [8]. PEH has been observed after aerobic exercise on both treadmill and cycloergometer, as well as after resistance exercise, in normotensive, hypertensive and diabetic hypertensive individuals [4,9–12]. However, many individuals with T2D present limitations such as diabetic foot ulcers that may compromise the performance of lower-limb exercises. An alternative for them would be arm-cranking exercise.

McDonald et al. [10] and Almeida et al. [11] reported PEH after an arm-cranking exercise bout in borderline hypertensive and healthy young individuals, respectively. Haddad et al. [5] found significant BP reductions in paraplegic individuals after a 12-week arm-cranking exercise training. However, to our knowledge, there are no studies in individuals with T2D, who would beneficiate from both acute and chronic BP adaptations.

Besides BP, rate-pressure product (RPP) and pulse pressure (PP) are also predictors of cardiovascular risk [13–16]. RPP indicates the metabolic demand imposed to the heart under different situations. PP reflects the interaction

between cardiac and vascular performances and is an indirect view of the ventricular-arterial coupling.

We aimed to investigate:

- the occurrence of PEH after a submaximal incremental test at baseline as well as after 4 weeks of arm-cranking exercise training;
- and the effects of this short-term intervention on resting BP, HR, RPP and PP in hypertensive type-2 diabetics.

We hypothesized that PEH would occur in response to both the first and the second incremental tests and that there would be a trend of reduction in resting BP, RPP and PP.

2. Methods

The procedures were approved by the Ethics Committee on Human Research from the State Health Secretary (SES/DF no. 050/2007) and were performed in conformity with the Declaration of Helsinki.

2.1. Sample

Thirty individuals with T2D and AH [16] were initially recruited from a Diabetic Health Care Program at the Catholic University Hospital. T2D and AH diagnosis were confirmed by a physician through a series of screening exams prior to the participants' admission in the Program. From these individuals, 20 met the inclusion criteria and volunteered to participate in the study.

The individuals were all sedentary, but performed 40 min of stretching exercises (physiotherapy) twice a week, as part of the Health Care Program, which also included blood glucose and blood pressure monitoring as well as informative talks. Their medical therapy – hypoglycemics (sulfonylurea), anti-diabetic drugs (metformin) and anti-hypertensive drugs (calcium channel antagonists and/or

diuretics) – was kept throughout the study and was similar in the two groups. The women were not under hormonal replacement therapy. A history of myocardial infarction or cerebral stroke, severe secondary complications such as blindness or wounded diabetic foot and physical or cardiovascular problems that could impair the accomplishment of exercise were considered as exclusion criteria.

The sample was randomly split into two groups: exercise trained (TRA; $n = 10$) and no exercise control (CON; $n = 10$). However, four individuals from the control group (two men) did not conclude the procedures because of personal ($n = 1$) or health-related problems: eye or leg surgery ($n = 2$); tumor diagnosis ($n = 1$).

2.2. Procedures

Before the first incremental test session, the individuals reported to the laboratory in a different day. On this occasion, they were explained about the procedures of the study and signed a written informed consent prior to participation. Afterwards, they underwent an anthropometric assessment (body mass, height and waist circumference), a resting electrocardiogram for cardiologic evaluation and also performed 15 min of arm-cranking exercise with no external workload to get familiarized with the exercise biomechanics.

At least 48 hours after the initial visit, both groups returned to the laboratory to perform the first submaximal incremental arm-cranking exercise test (IT-1). IT-1 results were considered as pre-training data. Then, group TRA performed 4 weeks of arm-cranking exercise training, while group CON kept only its regular daily activities. After this period, both groups performed a second submaximal incremental test (IT-2) for comparisons. IT-2 was performed at the same time of the day of IT-1 (in the afternoon) and the individuals were asked to ingest a similar meal 2 h before both tests.

2.3. Exercise protocols

2.3.1. Incremental Test (IT)

After arriving at the laboratory, the individuals rested for 20 min in a seated position. During this period, BP and heart rate (HR) were measured at each 5 min and a resting average was calculated. Both the IT-1 and IT-2 were performed on an arm ergometer at a cadence of 60 rpm. There was a 1-min warm-up with no external workload and then a workload of 6 W was added and increments of 6 W were applied at each 3 min of exercise until the individual achieved a HR corresponding to 85% of his/her predicted maximal HR, calculated through the formula “ $HR_{max} = 208 - (0.7 \times age)$ ” [17]. There was a 1-min interval between the stages. Immediately after the end of each stage, HR was recorded. Then, during the first 30 s of the resting interval, BP was measured. Afterwards, the rate of perceived exertion (RPE) was obtained and a blood sample was collected from the ear lobe for blood lactate analysis. The workload was also increased during the resting interval.

This IT protocol was previously shown to effectively induce hypotensive responses in young healthy individuals [11]. Changes in blood lactate concentration ($[lac]$) were used for calculating the lactate threshold (LT) on IT-1. The

HR corresponding to the LT was recorded and then used to establish the training intensity (90–100% LT) for group TRA. The RPE scores were obtained by using Borg’s 6 to 20 points [18] Scale. During the post-exercise recovery period, HR and BP were measured at each 15 min for 1 h.

2.3.2. Arm-cranking training sessions

For group TRA, the arm-cranking training comprised exercise sessions of 20 min, two times per week, for 4 weeks. The cadence was set at 60 rpm at an intensity corresponding to 90 to 100% LT, which meant a workload of 70 to 75% HR_{max} . HR was measured continuously (Polar F5, Kempele, Finland) during the training sessions to ensure the exercise intensity was kept within the established range. BP and blood glucose measurements were performed before and after exercise for safety reasons (data not shown).

2.4. Measurements

2.4.1. Blood pressure (BP)

During the resting and recovery periods, the individuals remained seated in a quiet room without noise. BP was measured one time for each point, always on the left arm positioned at heart level, using an oscillometric sphygmomanometer (BP 3AC1-1, Microlife, Berneck, Switzerland). During IT-1 and IT-2, BP was measured through auscultatory method, using an analogical sphygmomanometer (Tycos, SP, Brasil) and a stethoscope (Becton Dickinson, NJ, USA) only for monitoring purposes. Mean arterial pressure (MAP) was calculated as $MAP = [(SBP - DBP) \times 0.33 + DBP]$ and PP was calculated as $PP = SBP - DBP$. RPP was calculated as $RPP = SBP \text{ (mmHg)} \times \text{Heart Rate (bpm)}$.

Table 1 Characteristics of the participants ($n = 16$).

	Trained group ($n = 10$)	Control group ($n = 6$)
Men/Women	6/4	4/2
Age (years)	61 ± 9	54 ± 5
Time of T2D diagnosis (years)	13 ± 10	14 ± 8
Body mass (kg)	71 ± 13	67 ± 10
Height (m)	1.6 ± 0.1	1.6 ± 0.1
Body mass index (kg/m ²)	27 ± 4	26 ± 2
Resting systolic blood pressure (mmHg) ^a	139 ± 19	133 ± 15
Resting diastolic blood pressure (mmHg) ^a	78 ± 10	79 ± 12
Resting heart rate (bpm)	69 ± 14	78 ± 10
Maximal predicted heart rate (bpm) [24]	165 ± 6	170 ± 3
Waist circumference (cm)	92 ± 14	88 ± 9
Resting blood glucose (mg/dL) ^a	160 ± 46	166 ± 28

T2D: type-2 diabetes mellitus.

^a Blood pressure and blood glucose were measured at resting and the individuals were under the effect of their regular medication and at a non-fasting state.

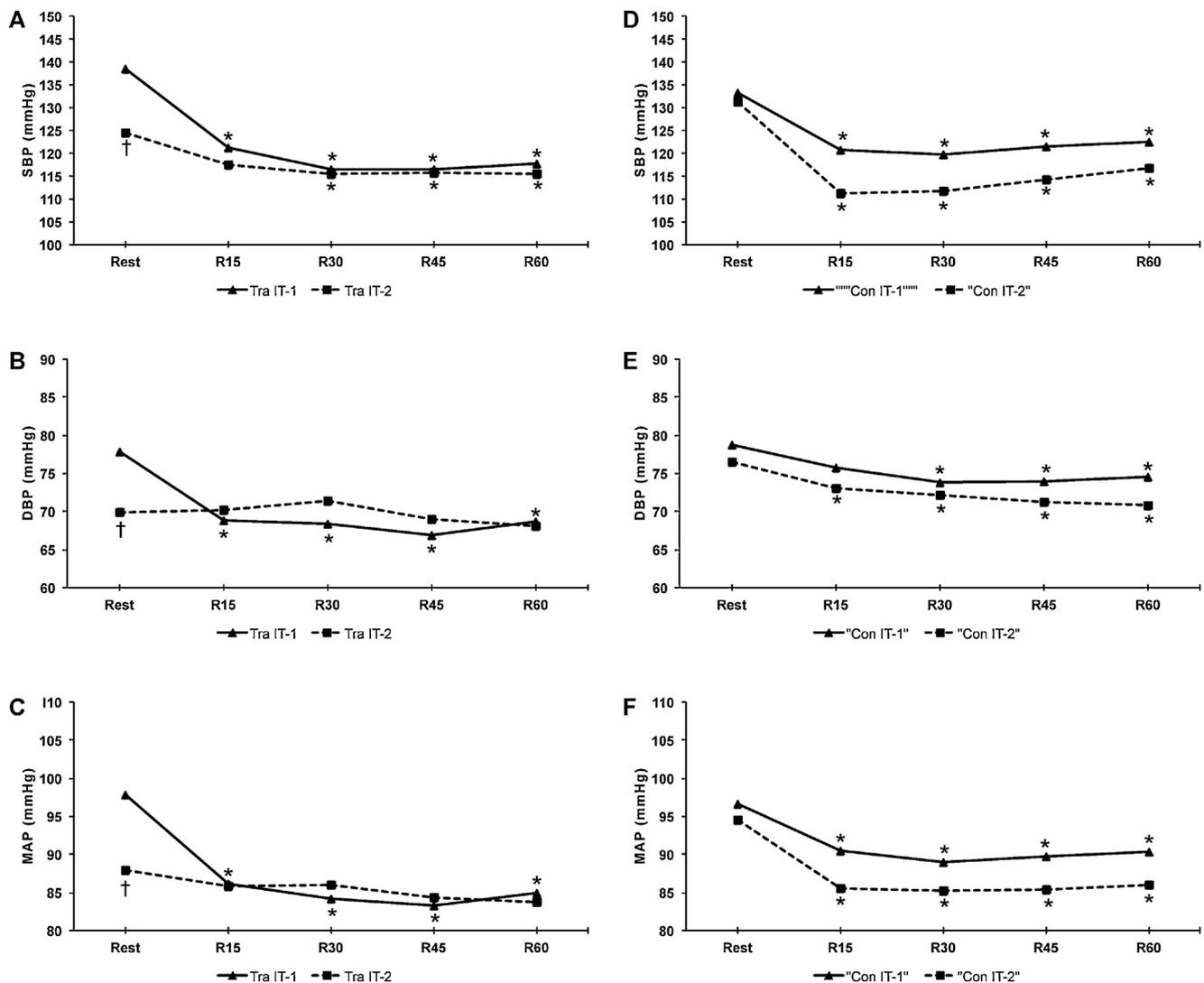


Figure 1 Systolic blood pressure (SBP), diastolic blood pressure (DBP) and mean arterial pressure (MAP) at pre-exercise resting (Rest) and for 1-hour of post-exercise recovery (R15-R60) after incremental test 1 (IT-1) and 2 (IT-2) for the trained (TRA; panels A, B and C) and control (CON; panels D, E and F) groups. * $P \leq 0.05$ compared to Rest. † $P \leq 0.05$ compared to IT-1.

2.4.2. Blood glucose and blood lactate

25 μ l blood samples were taken from the ear lobe through heparinized capillary tubes and deposited in Eppendorf micro-tubes containing 50 μ l of Sodium Fluoride (NaF) at 1%. The samples were kept in ice until the moment of analyses. Blood lactate and glucose concentrations were determined electroenzymatically (Yellow Springs Instruments 2.700 STAT, Ohio, USA).

2.4.3. Data analysis

Descriptive statistics were performed and the data is presented as mean \pm standard deviation. Student's *t*-test was applied to analyze the occurrence of PEH and two-way ANOVA with an adjustment of Bonferroni was applied to look for pre- and post-training as well as between-group differences. The level of significance was set at $P \leq 0.05$ and all analyses were performed using Statistica 7.0 (Statsoft, Oklahoma, USA).

3. Results

The descriptive characteristics of the participants at baseline are presented in Table 1 and did not differ between the trained and control groups ($P > 0.05$).

3.1. Acute arm-cranking exercise effects

As hypothesized, both groups presented significant PEH ($P \leq 0.05$) of SBP, DBP and MAP throughout 1 h following the pre-training incremental arm-cranking exercise test (IT-1) (Fig. 1). However, for the group that underwent the exercise training intervention (TRA), the hypotensive effects of an acute bout of arm-cranking exercise were attenuated following the post-training incremental test (IT-2). This response was significant for SBP, MAP and more markedly for DBP (Fig. 1A–F). The control group presented similar PEH patterns for SBP, DBP and MAP following IT-2 when compared to those following IT-1.

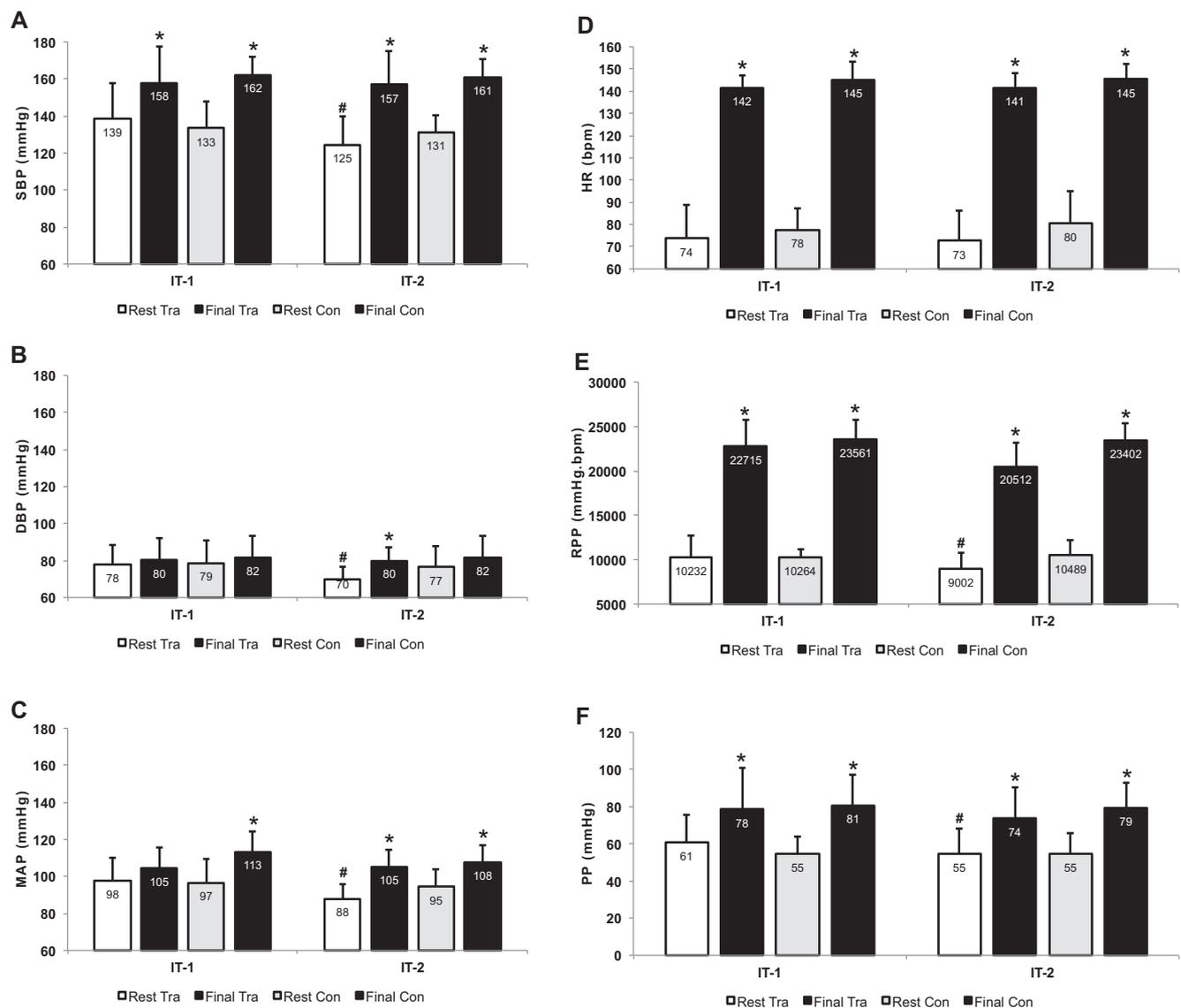


Figure 2 Systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP), heart rate (HR), rate-pressure product (RPP) and pulse pressure (PP) at pre-exercise resting (Rest) and at the end (Final) of incremental test 1 (IT-1) and 2 (IT-2) for the trained (TRA) and control (CON) groups. * $P \leq 0.05$ compared to Rest; # $P \leq 0.05$ compared to IT-1.

3.2. Arm-cranking exercise training effects

With regards to the resting parameters, after 4 weeks of arm-cranking exercise training, there were observed reductions in BP, RPP and PP ($P \leq 0.05$) for group TRA, whereas for group CON there were no significant differences after the same period of time without exercise (Fig. 2).

After the 4-week intervention, significant differences in the final workload attained at the IT and total exercise duration were observed only for group TRA (Table 2). Blood lactate concentration was not different in spite of the increase in the final workload.

4. Discussion

The main findings of the present study were that an acute arm-cranking exercise session was effective in eliciting PEH

and that this response was attenuated after 4 weeks of exercise training. This short-term intervention also evoked reductions in resting BP, PP and RPP in individuals with T2D and AH.

As hypothesized, PEH occurred for both groups in the baseline non-trained condition (IT-1). This is in agreement with previous studies that have demonstrated PEH of SBP and MAP in individuals with T2D after a single bout of cycling [4,19] or treadmill exercise [9] as well as PEH of SBP, MAP and DBP after an acute bout or arm-cranking exercise both in healthy [11] and borderline hypertensive individuals [10].

For being an unconventional modality and activating a smaller muscle mass, arm-cranking exercise may induce higher metabolic and hemodynamic stress that can be related to vasodilation [20,21] and this is likely to influence the changes in DBP, which represents the efficiency of vasodilatory mechanisms [22].

Table 2 Final workload, exercise duration, blood lactate concentration, blood pressure and heart rate at the end of IT-1 and IT-2.

	Trained group		Control group	
	IT-1	IT-2	IT-1	IT-2
Workload (W)	23 ± 8	30 ± 9 ^{*,**}	21 ± 9	19 ± 7
Exercise duration ^a (min)	15 ± 6	20 ± 6 ^{*,**}	14 ± 7	13 ± 5
bLac concentration (mmol/L)	5.7 ± 0.1	5.1 ± 0.3	6.2 ± 0.1	5.1 ± 0.1
Systolic blood pressure (mmHg)	158 ± 19	157 ± 18	162 ± 10	161 ± 10
Diastolic blood pressure (mmHg)	80 ± 11	80 ± 7	82 ± 12	82 ± 12
Heart rate (bpm)	142 ± 5	141 ± 7	145 ± 8	145 ± 7

* $P \leq 0.05$ compared to IT-1; ** $P \leq 0.05$ compared to control group. bLac: blood lactate.

^a Including one-minute intervals between incremental stages.

The magnitude of PEH of SPB and MAP observed in the present study (Fig. 1) are comparable to those reported in the studies of Lima et al. [4] after 20 min of cycloergometry at intensities corresponding to 90% and 110% of LT, and Cunha et al. [9] after treadmill exercise.

After IT-2, the group CON presented a similar PEH response as in IT-1, for SBP, DBP and MAP, while this response was attenuated for group TRA, especially regarding DBP and MAP. Our hypothesis was that PEH would occur for the two groups on both IT-1 and IT-2. However, there is evidence that the magnitude of PEH is influenced by pre-exercise resting BP values, being higher in individuals with higher pre-exercise BP [23]. These results are in agreement with those from Moraes et al. [24] who showed an attenuation of the hypotensive effect of acute resistance exercise after 12 weeks of moderate-intensity resistance exercise training in hypertensive individuals. Similarly to the present study, the authors found a significant reduction in resting BP after training. This response may, at least in part, account for the reductions in the magnitude of PEH.

The significant reductions in resting BP, PP and RPP were beyond our expectations, due to the short-term characteristic of the intervention. In hypertensive individuals, Monteiro et al. [25] showed similar reductions in BP after two months of aerobic training. It is suggested that the accumulation of the acute hypotensive effects over regular exercise training may lead to benefit chronic adaptations [26].

Reductions in RPP and PP are cardioprotective as well. Reductions in RPP suggest a lower cardiac overload [13] and a lower PP (below 50 mmHg) indicates a reduced risk of cardiovascular complications and all-cause mortality. In the present study, although PP values remained around 55 mmHg for group TRA after the 4-week exercise training intervention, there was a significant ($P < 0.05$) reduction in relation to pre-training values (61 mmHg), with no changes for group CON. This beneficial effect of exercise training on PP may indicate more a vascular than a central effect. Our hypothesis is that a longer-term exercise period should promote further benefits.

The arm-cranking training was performed at an intensity correspondent to the LT, which represents a moderate exercise (70 to 75% of HR_{max}) of low metabolic stress [27]. This intensity was well borne by the participants, as indicated by RPE scores around 12 to 13 points on the Borg's Scale [18] during the training sessions for group TRA.

On IT-2, group TRA attained higher exercise duration and final absolute workload when compared to IT-1, despite of the individuals reaching the same percentage of predicted HR_{max} (85%; Table 2). This suggests an improvement in the aerobic capacity for group TRA, while there were no changes for group CON.

Throughout the study, both groups kept their regular T2D and AH medical therapy and also continued their participation in the Diabetes Health Care Program, which included stretching exercises twice a week. This indicates that the observed training effects were a consequence of the arm-cranking intervention, as group CON showed no alterations. Also, it suggests that the benefits of regular physical exercise may be additional to pharmacological therapy and reinforces the importance of a multidisciplinary approach when seeking for a better hemodynamic control in individuals with T2D and AH. Thus, the present results suggest that arm-cranking exercise training is safe and may benefit these individuals with regards to BP control.

One limitation of the present study was the small sample size, in part because of the individuals who dropped out. However, use of a control group reinforces the observed exercise effects. A second limitation regards the short duration of intervention. In spite of this, the arm-cranking exercise training evoked positive changes in resting hemodynamic parameters. However, we suggest additional studies with long-term interventions to confirm the present results. Future studies should also analyze bigger samples including individuals with more severe complications, such as foot ulcers.

5. Conclusion

There was observed PEH of SBP, DBP and MAP after a submaximal incremental arm-cranking exercise performed by sedentary T2D individuals with AH. After 4 weeks of arm-cranking exercise training, this hypotensive effect was attenuated, with a significant PEH only of SBP. This attenuation may be related to the lower resting BP values observed at IT2 for group TRA. Lower resting RPP and PP values were also observed for the trained group after the 4-week period. These results suggest that arm-cranking exercise is effective to promote PEH in type-2 diabetics with hypertension.

Disclosure of interest

The authors declare that they have no conflicts of interest concerning this article.

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References

- [1] Oliveira PJ. Cardiac mitochondrial alterations observed in hyperglycaemic rats—what can we learn from cell biology? *Curr Diabetes Rev* 2005;1:11–21.
- [2] Khullar M, Al-Shudiefat AAS, Ludke A, Binepal G, Singal PK. Oxidative stress: a key contributor to diabetic cardiomyopathy. *Can J Physiol Pharmacol* 2010;88:233–40.
- [3] de Pinho RA, Araújo MC, Ghisi GLM, Benetti M. Coronary heart disease, physical exercise and oxidative stress. *Arq Bras Cardiol* 2010;94:549–55.
- [4] Lima LCJ, Assis GV, Hiyane W, Almeida WS, Arsa G, Baldissera V, et al. Hypotensive effects of exercise performed around anaerobic threshold in type 2 diabetic patients. *Diabetes Res Clin Pract* 2008;81:216–22.
- [5] Haddad S, Silva PRS, Barretto ACP, Ferrareto I. Efeito do Treinamento Físico de Membros Superiores Aeróbio de Curta Duração no Deficiente Físico com Hipertensão Leve. *Arq Bras Cardiol* 1997;69:169–73.
- [6] Monteiro LZ, Fiani CRV, Freitas MCF, Zanetti ML, Foss MC. Decrease in blood pressure, body mass index and glycemia after aerobic training in elderly women with type 2 diabetes. *Arq Bras Cardiol* 2010;95:563–70.
- [7] Kenney MJ, Seals DR. Post-exercise hypotension: key features, mechanisms and clinical significance. *Hypertension* 1993;22:653–64.
- [8] Park S, Jastremski CA, Wallace JP. Time of day for exercise on blood pressure reduction in dipping and non-dipping hypertension. *J Hum Hypertens* 2005;19:597–605.
- [9] Cunha GA, Rios ACS, Moreno JR, Braga PL, Campbell CSG, Simões HG, et al. Hipotensão pós-exercício em hipertensos submetidos ao exercício aeróbio de intensidades variadas e exercício de intensidade constante. *Rev Bras Med Esporte* 2006;12:313–7.
- [10] MacDonald JR, MacDougall JD, Hogben CD. The effects of exercising muscle mass on post-exercise hypotension. *J Hum Hypertens* 2000;14:317–20.
- [11] Almeida WS, Lima LCJ, Cunha RR, Simões HG, Nakamura FY, Campbell CSG. Post-exercise blood pressure responses to cycle and arm-cranking. *Sci Sports* 2010;25:74–80.
- [12] Lizardo JHF, Modesto LK, Campbell CSG, Simões HG. Post-exercise hypotension: comparison between different intensities of exercise on a treadmill and a cycle ergometer. *Rev Bras Cineantropom Des Hum* 2007;9:115–20.
- [13] Hui SC, Jackson AS, Wier LT. Development of normative values for resting and exercise rate pressure product. *Med Sci Sports Exerc* 2000;32:1520–7.
- [14] Blacher J, Staessen JA, Girerd X, Gasowski J, Thijs L. Pulse pressure not mean pressure determines cardiovascular risk in older hypertensive patients. *Arch Intern Med* 2000;160:1085–9.
- [15] de Sousa MA, Hermann JLV, Guimarães JB, Menezes PPO, Carvalho ACC. Evaluation of systolic, diastolic and pulse pressure as risk factors for severe coronary arteriosclerotic disease in women with unstable angina non ST-elevation acute myocardial infarction. *Arq Bras Cardiol* 2004;82:426–9.
- [16] Chobanian AV, Bakris GL, Black HR, Cushman WC, Green LA, Izzo Jr JL, et al. National high blood pressure education program coordinating committee. The seventh report of the joint national committee on prevention, evaluation and treatment of high blood pressure. *Hypertension* 2003;42:1206–52.
- [17] Tanaka H, Monahan KD, Seals DR. Age-predicted maximal heart rate revisited. *J Am Coll Cardiol* 2001;37:153–6.
- [18] Borg GAV. Psychophysical bases of perceived exertion. *Med Sci Sports Exerc* 1982;14:377–81.
- [19] Motta DF, Lima LCJ, Arsa G, Russo PS, Almeida WS, Sales MM, et al. Effect of type 2 diabetes on plasma kallikrein activity after physical exercise and its relationship to post-exercise hypotension. *Diabetes Metab* 2010;36:363–8.
- [20] Lyons S, Richardson M, Bishop P, Smith J, Heath H, Giesen J. Excess post-exercise oxygen consumption in untrained men following exercise of equal energy expenditure: comparisons of upper and lower body exercise. *Diabetes Obes Metab* 2007;9:889–94.
- [21] Volianitis S, Yoshiga CC, Nissen P, Secher NH. Effect of fitness on arm vascular and metabolic responses to upper body exercise. *Am J Physiol Heart Circ Physiol* 2004;286:H1736–41.
- [22] Bermudes AMLM, Vassallo DV, Vasquez EC, Lima EG. Ambulatory blood pressure monitoring in normotensive individuals undergoing two single exercise sessions. Resistive exercise and aerobic exercise. *Arq Bras Cardiol* 2003;82:65–71.
- [23] Cornelissen VA, Fagard RH. Exercise intensity and postexercise hypotension. *J Hypertens* 2004;22:1859–61.
- [24] Moraes MR, Bacurau RFP, Simões HG, Campbell CSG, Pudo MA, Wasinski F, et al. Effect of 12 weeks of resistance exercise on post-exercise hypotension in stage 1 hypertensive individuals. *J Hum Hypertens* 2011;1:1–7.
- [25] Monteiro HL, Rolim LMC, Squinca DA, Silva FC, Ticianeli CCC, Amaral SL. Efetividade de um programa de exercícios no condicionamento físico, perfil metabólico e pressão arterial de pacientes hipertensos. *Rev Bras Med Esporte* 2007;13:107–12.
- [26] Polito MD, Farinatti PTV. Comportamento da pressão arterial após exercícios contra-resistência: uma revisão sistemática sobre variáveis determinantes e possíveis mecanismos. *Rev Bras Med Esporte* 2006;12:386–92.
- [27] Meyer T, Görge G, Schwaab B, Hildebrandt K, Walldorf J, Schäfer C, et al. An alternative approach for exercise prescription and efficacy testing in patients with chronic heart failure: a randomized controlled training study. *Am Heart J* 2005;149:926e1–7.