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Original Article

Effects of aerobic exercise intensity on 24-h ambulatory blood pressure in individuals with type 2 diabetes and prehypertension

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Abstract. [Purpose] To verify the effects of different intensities of aerobic exercise on 24-hour ambulatory blood pressure (BP) responses in individuals with type 2 diabetes mellitus (T2D) and prehypertension. [Subjects and Methods] Ten individuals with T2D and prehypertension (55.8 ± 7.7 years old; blood glucose 133.0 ± 36.7 mg·dL^{-1} and awake BP 130.6 ± 1.6/ 80.5 ± 1.8 mmHg) completed three randomly assigned experiments: non-exercise control (CON) and exercise at moderate (MOD) and maximal (MAX) intensities. Heart rate (HR), BP, blood lactate concentrations ([Lac]), oxygen uptake (VO_2), and rate of perceived exertion (RPE) were measured at rest, during the experimental sessions, and during the 60 min recovery period. After this period, ambulatory blood pressure was monitored for 24 h. [Results] The results indicate that [Lac] (MAX: 6.7±2.0 vs. MOD: 3.8±1.2 mM), RPE (MAX: 19±1.3 vs. MOD: 11±2.3) and VO_2peak (MAX: 20.2±4.1 vs. MOD: 14.0±3.0 mL·kg^{-1}·min^{-1}) were highest following the MAX session. Compared with CON, only MAX elicited post-exercise BP reduction that lasted for 8 h after exercise and during sleep. [Conclusion] A single session of aerobic exercise resulted in 24 h BP reductions in individuals with T2D, especially while sleeping, and this reduction seems to be dependent on the intensity of the exercise performed.

Key words: Metabolic disease, Systemic arterial hypertension, lactate threshold

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INTRODUCTION

Type 2 diabetes mellitus (T2D) is a metabolic disorder characterized by the inability of insulin to properly perform its function, resulting in chronic hyperglycemia[1)] and it is also associated with endothelial dysfunction[2)], increase in sympathetic tone[3)] and several cardiovascular disorders, such as systemic arterial hypertension (SAH)[4)] and coronary artery disease (CAD)[5)]. The increase in the incidence of T2D is mainly the result of genetic predisposition, dietary excess of foods rich in fat and sugar, obesity, sedentarism and low levels of physical fitness[6, 7)].

The frequent practice of physical exercise helps to both prevent and treat T2D and its complications[8–10)]. The benefits of exercise for diabetic individuals include better glycemic control[11, 12)], reduction of blood pressure (BP)[13–15)] and improvement of cardiorespiratory fitness[12, 16)] all of which, are associated with cardiovascular events and mortality[17)]. The reduction of BP in the post-exercise (recovery) period to values below those observed during pre-exercise (rest) is called post-exercise hypotension (PEH)[18)]. This phenomenon can be observed both in normotensive[19)] and hypertensive individuals[20, 21)], and in the latter, a post-exercise BP reduction from 12 h[22, 23)] to 24 h[24, 25)] has also been observed.

The prescription of an appropriate amount of exercise is recommended for T2D patients, and it should be based on intensities that are related to their aerobic and physical capacities. To accomplish this, it is necessary to perform incremental tests for the evaluation of functional ability, such as the anaerobic threshold (AT) and maximum oxygen uptake (VO_2max) tests[26–28)]. These tests are currently used by studies investigating the prescription of aerobic exercise for BP control in individuals with T2D[14, 15)].

However, some studies[14, 15)] have only analyzed BP for two hours post-exercise and have not investigated whether the benefits extend over the following 24 hours, including sleep. It is known that T2D is associated with endothelial dysfunction[2)], therefore, this pathology could attenuate the expected benefits of acute aerobic exercise on BP[3)]. Consequently, the objectives of this study were to verify and compare: 1) the effects of a single aerobic exercise session on 24-hour BP responses and 2) the effects of aerobic exercise

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intensity on 24-hour BP responses of individuals with T2D and prehypertension.

SUBJECTS AND METHODS

After approval by the local human research ethics committee (CEP of. 013/2008), 10 participants aged between 45 and 70 years old were enrolled (3 men and 7 women; Table 1) after they signed an informed consent form before participation in the study. Inclusion criteria were sedentary life style, a diagnosis of T2D for at least one year, glycemic control through nutrition and/or medication (e.g. sulfonylureas, metformin, metformin+glibenclamide, glimepiride, pioglitazone chloridrate), no use of exogenous insulin and no chronic complications, such as, diabetic foot, nephropathy, retinopathy, neuropathies or cardiovascular diseases. All participants were recruited from a Public Hospital at Taguatinga, and all had been previously assessed in standard tests conducted by endocrinologists and cardiologists. This meant that, at the time of recruitment, all subjects with a positive history for any of the tests were eliminated from the study.

After anthropometric and resting electrocardiography (ECG) evaluations and familiarization with the experimental procedures, the volunteers participated in three cardiologist-supervised experimental sessions on different days: 1) maximal exercise (MAX); 2) moderate exercise (MOD); and 3) a control session (CON) with no exercise. Sessions 2 and 3 were performed on alternate days in a randomized order at the same time of day (between 8:30 and 9:00 h), with a 48-hour interval between sessions. Two hours before the sessions the participants ate a standardized breakfast of 315 kcal, containing 66% (51.6 g) carbohydrates, 6% (4.6 g) of protein and 27% (9.5 g) of fat.

Before sessions the participants remained at rest for 20 minutes for BP (Dyna-MAPA − Cardios®) and HR (Polar® S810i) measurements, while capillary blood samples were collected for [Lac] determination (YSI 2700 − Yellow Springs − USA). During the post-experimental period, the participants remained seated for a period of 60 minutes, while the measurements of BP and HR, and blood sample collections were taken every 15 minutes. After that, the participants were allowed 20 minutes for personal hygiene before the placement of an ambulatory blood pressure monitor (Dyna-MAPA − Cardios®), which measured their BP for the 24 hours subsequent to their exercise or control sessions. Ambulatory blood pressure monitor (ABPM) was carried out every 30 min after the sessions until 2300 h, after that time, hourly, until 0700 h, as per the IV Guidelines For Ambulatory Blood Pressure Monitoring[28]. ABPM data were considered in the analysis when ≥90% of the measurements were valid. Participants were instructed to perform their habitual daily activities and to keep the non-dominant upper limb (where the measuring cuff was placed) in a relaxed position while the BP was being measured[28].

MAX consisted of a graded cardiopulmonary exercise stress test (Lode Excalibur Sports, Netherlands). VO_2peak was determined by breath-by-breath analysis of expiratory gases during exercise as previously described[29]. The gas analyzer was calibrated with a 3-L syringe (flow calibration) and a standard mixture of gas containing 4.9% CO_2 and 17% O_2 (gas calibration). An incremental load test was initiated with one minute of warm-up at a zero Watt (W) load, followed by increments of 15 W every 3 minutes, consisting of continuous cycling at a constant cadence (60 revolutions per minute) until voluntary exhaustion of the participant. At rest and during the final 10 sec of each stage of the MAX, blood samples were collected for blood lactate analysis ([Lac]) using a YSI 2700 biochemistry analyzer (YSI Inc, Yellow Springs, OH, USA). In addition, BP was measured by the auscultatory method, using a mercury column sphygmomanometer (Tycos Instrumentos Hospitalares, São Paulo, Brazil). Heart rate (HR) was monitored using a Polar® S810i heart-rate monitor (Polar Electro Oy, Kempele, Finland), rate of perceived exertion (RPE) was rated on the 20-point Borg scale[30], and ventilatory variables were also recorded (MetaLyzer 3B System; Cortex, Leipzig, Germany). MAX allowed identification of the lactate threshold (LT) and measurement of the VO_2peak. The LT was identified through visual inspection of the lactate curve; the LT was considered the intensity at which a loss of linearity and an abrupt and exponential increase in the lactate curve occurred[31, 32].

The MOD session consisted of 20 minutes of cycle ergometer exercise (Lode Excalibur Sports, Netherlands) at a constant load corresponding to 90% of the LT (90%LT). The CON was a 20-minute session of seated rest. At 10 and 20 min during MOD and CON, BP, HR, [Lac], RPE and ventilatory variables were measured.

Table 1. Anthropometric characteristics and parameters of aerobic fitness during incremental load test performed by individuals with T2D (n=10)

Age (years)	55.8 ± 7.7
Weight (kg)	79.4 ± 14.0
Height (cm)	160.7 ± 11.3
BMI ($kg \cdot m^{-2}$)	30.7 ± 4.2
Abdominal circumference (cm)	102.1 ± 13.7
BodyFat (%)	29.2 ± 9.9
Time with DM (years)	6.3 ± 3.1
FG ($mg \cdot dL^{-1}$)	133.0 ± 36.7
Awake SBP (mmHg)	130.6 ± 1.6
Awake DBP (mmHg)	80.5 ± 1.8
HRmax (bpm)	150.3 ± 23.5
Pmax (Watts)	85.5 ± 22.4
[Lac] max (mM)	6.7 ± 2.0
VO_2peak ($ml \cdot kg^{-1} \cdot min^{-1}$)	20.2 ± 4.1
LT (watts)	49.5 ± 23.5
[Lac] LT (mM)	3.2 ± 0.9
VO_2 at LT ($ml \cdot kg^{-1} \cdot min^{-1}$)	14.6 ± 3.7

BMI: body mass index; FG: fasting glycemia; SBP: systolic blood pressure; DBP: diastolic blood pressure; VO_{2peak}: peak consumption of oxygen; Pmax: the highest power output (WATTS) obtained in the incremental test on a cycle ergometer; HRMax: maximum heart rate; [Lac]: blood lactate concentration; LT: lactate threshold

Table 2. Mean (±DP) results of metabolic and hemodynamic parameters, rate of perceived exertion and the duration of sessions

	MAX	MOD	CON
[Lac] (mM)	6.7 ± 2.0 †	3.8 ± 1.2	2.5 ± 0.8*
VO_2 ($mL \cdot kg^{-1} \cdot min^{-1}$)	20.2 ± 4.1†	14.0 ± 3.0	4.2 ± 0.3*
% VO_{2peak}	100 ± 0.0 †	69.4 ± 8.1	-
Watts	85.5 ± 22.4†	44.6 ± 21.2	-
HR (bpm)	150.3 ± 23.5†	114.5 ± 25.1	73.2 ± 9.1*
SBP (mmHg)	197.8 ± 14.0†	147.0 ± 15.5	125.2 ± 13.3*
DBP (mmHg)	89.7 ± 6.2	84.5 ± 7.2	78.8 ± 7.3
RPE (score)	19 ± 1.3 †	11 ± 2.3	-
Duration (min)	18.7 ± 4.5	20.0 ± 0.0	20.0 ± 0.0

[Lac]: concentration of blood lactate; Peak VO_2: highest value of oxygen consumption; $\%VO_{2\ peak}$: percentage of peak oxygen consumption reached; HR: heart rate; SBP: systolic blood pressure; DBP: diastolic blood pressure; RPE: rate of perceived exertion. * $p \leq 0.05$ in relation to sessions MAX and MOD; † $p \leq 0.05$ in relation to MOD

Table 3. Mean results (±SD) of 24-h systolic blood pressure (SBP), diastolic (DBP) and mean arterial pressure (MAP) after the MAX, MOD and CON sessions

Parameters		MAX	MOD	CON
SBP (mmHg)	Resting	132.1± 11.4	129.0 ± 10.5	130.8 ± 8.4
	24 h	124.3 ± 7.4 *	127.7 ± 7.1	132.8 ± 10.6
	Waking	126.6 ± 8.0 *	129.7 ± 7.1	135.8 ± 10.9
	Sleeping	116.8 ± 9.8 †#	121.5 ± 12.0	124.6 ± 11.6
DBP (mmHg)	Resting	81.6 ± 6.7	80.3 ± 8.8	79.7 ± 10.1
	24 h	76.3 ± 4.5 *†	78.9 ± 3.7	82.2 ± 5.0
	Waking	77.9 ± 5.1 *	80.4 ± 3.8	83.9 ± 5.2
	Sleeping	70.8 ± 4.7*†#‡	73.6 ± 7.4	77.1 ± 5.4
MAP (mmHg)	Resting	102.6 ± 8.2	101.0 ± 8.4	101.3 ± 9.3
	24 h	95.4 ± 4.6 *†	98.2 ± 3.4	102.5 ± 6.9
	Waking	97.3 ± 5.5 *	100.2 ± 3.5	104.8 ± 7.4
	Sleeping	89.2 ± 5.2*†#‡	91.6 ± 6.9 †	95.8 ± 6.3

*$p<0.05$ in relation to control session; †$p<0.05$ in relation to pre-exercise rest of the same session; # in relation to waking period of the same session; ‡ in relation to 24 h of the same session

Statistical analyses

Data are presented as mean (±) standard deviation and as absolute variations (delta absolute), as indicated for each analysis. After assessing the normality and homogeneity of the data using the Shapiro-Wilk test and Levene's test, respectively, between- and within-groups comparisons were performed using Split-Plot ANOVA (Mixed ANOVA) with Scheffe's post hoc test. When any of the dependent variables did not show sphericity in the Mauchly's test, the epsilon of Greenhouse-Geisser was used to analyze the F statistic. The level of significance was chosen as 5% ($p \leq 0.05$) and all analyses were carried out using the Statistical Package for the Social Sciences (SPSS) 15.0.

RESULTS

The subjects were 10 persons, 3 men and 7 women with T2D, according to the fasting glycemia[1)], and prehypertension[33)]. The participants were overweight to obese[34)] and had low physical fitness[35)] (Table 1). The aerobic fitness, metabolic and hemodynamic parameters obtained during all the sessions (MAX, MOD and CON) are presented in Tables 1 and 2. No subject exhibited a hypertensive response, defined as SBP ≥210 mmHg and DBP ≥ 105 mmHg[36)], to the graded exercise test.

Table 3 presents the mean values of SBP, DBP and MAP at rest and during ambulatory measurement of blood pressure (ABPM) after the experimental sessions and control. The post-MAX exercise SBP, DBP and MAP over 24 h, in the waking and sleeping (DBP and MAP) period were significantly different from the respective values obtained after the CON session ($p<0.05$).

Regarding the absolute changes (delta) of SBP, DBP and MAP during the 24 hours after the experimental sessions. A reduction in SBP was observed in the periods 0–2 h (−4.8 mmHg), 4–6 h (−7.6 mmHg), 6–8 h (−11.6 mmHg) post-MAX compared to CON (0–2 h: +5.6; 4–6 h: +6.3; 6–8 h: +9.1; 20–22: −1.4 mmHg), and in the period 20–22 h (−16.1

mmHg) compared to the CON (−1.4 mmHg) and MOD (−2.1 mmHg) ($p<0.05$). Furthermore, a reduction in SBP occurred between 16–22 h (16–18 h: −19.7 mmHg; 18–20 h: −17.6 mmHg; 20–22 h: −16.1 mmHg) after MAX compared to pre-exercise rest ($p<0.05$).

Significant reductions in DBP were observed from 0–2 h (−1.9 mmHg), 6–8 h (−5.8 mmHg), and 18–22 h (18–20 h: 12.2 mmHg; 20–22 h: −7.5 mmHg) post-MAX compared to CON (0–2 h: +7.6; and 6–8 h: +5.0; 18–20 h: −3.7; 20–22 h: +2.4 mmHg) ($p<0.05$). A nocturnal reduction in DBP between 16 and 20 h (16–18: −14.5; 18–20 h: −12.2 mmHg) was observed only after MAX ($p<0.05$).

Similarly the MAP, significant reductions were observed post-MAX when compared to CON in the periods 0–2 h (MAX: −3.9 vs. CON: +6.6 mmHg), 6–8 h (MAX: −7.9 vs. CON: +4.6 mmHg), and 18–22 h [MAX (18–20 h): −17.1 vs. CON: −7.0 mmHg; MAX (20–22 h): −10.4 vs. CON: +0.5 mmHg]. Between 14 and 22 h (14–16 h: −11.2; 16–18 h: −16.4; 18–20 h: −17.1; 20–22 h: −10.4 mmHg) there was a post-MAX reduction in MAP, as well as between 16 and 20 h (16–18 h: 12.1; 18–20 h: −11.0 mmHg) post-MOD compared to pre-exercise values ($p<0.05$).

No significant differences were found for SBP, DBP and MAP after the MOD session compared to after the MAX and CON sessions during the 24 h ABPM ($p>0.05$).

DISCUSSION

The present study investigated the effects of different exercise intensities on 24 hour BP responses in patients with T2D and prehypertension. The results reveal that, when compared to the day in which no exercise was performed (CON), the MAX session elicited a reduction in SBP, DBP and MAP over a 24 h period. Furthermore, the MAX session produced a significant reduction in pressure variation during sleep compared to the CON session, and also for SBP when compared to the MOD session (Table 3).

The majority of studies have investigated the effects of different exercise intensities on 24-hour BP responses in healthy and hypertensive subjects[21, 24]. Little research of this type has investigated individuals with T2D[15, 16], and when it has, the authors only analyzed BP for 2 h post-exercise. Therefore, little is known about the effects of different exercise intensities on non-pharmacologic control of 24 h BP in T2D individuals. In the present study, both the MAX and MOD sessions elicited significant BP reductions, suggesting that about 20 minutes of exercise has significant benefits. The results also suggest that the intensity of the exercise and the physiological stress generated play important roles in lowering the blood pressure of individuals with T2D.

Evidence has been presented that exercise intensity influences the BP response of diabetic and hypertensive individuals[14, 21]. However, Pescatello et al.[22] investigated the effects of exercise intensity (40 and 70% VO_2max) on the BP response of normotensive and hypertensive individuals, and found that intensity did not affect post-exercise BP reduction, which lasted for 12 hours regardless of the intensity, but only in the hypertensive group. Nevertheless, it is possible that the studied intensities (40 and 70% of VO_2max), both within in the same intensity domain (moderate-below the LT), masked the effect of exercise intensity.

Eicher et al.[24] conducted three experimental sessions of low intensity exercise (40% of VO_2peak), moderate intensity exercise (60% of VO_2peak) and vigorous intensity exercise (100% of VO_2peak), with 45 hypertensive men. They reported that PEH was proporcionate to the level of effort in the exercise sessions: the vigorous session caused the greatest reductions in SBP (−1.0 ± 1.7 mmHg) and DBP (−6.3 ± 1.2 mmHg) compared to the control session (10.7 ± 1.3 and −1.4 ± 0.9 mmHg). They also reported that for each 10% increase in VO_2peak there was a 1.5 mmHg and 0.6 mmHg decrease for SBP and DBP, respectively.

One of the main features of the present study was that T2D patient results were recorded for 24 hours, and that, compared to CON, a greater reduction in SBP occurred during the post-MAX sleeping period than during post-MOD. This is important for individuals with T2D, considering that elevated BP values during the day associated with a low nocturnal decrease (less than 10%) are factors that, in association with hyperglycemia, increase the risk of cardiovascular dysfunction. Jones et al.[37] studied 6 normotensive individuals who performed aerobic exercises at 40% and 70% VO_2max in the morning, and observed a MAP reduction only during sleep after 70% VO_2max exercise, which suggests that the exercise intensity could have been responsible for this result.

The difference in exercise intensity in this study (MAX and MOD) can be confirmed by the different levels of metabolic, hemodynamic, and cardiorespiratory stress, and by the perceptual responses collected during the experimental sessions. Significant differences were observed in the responses to MAX, with higher values of VO_2peak, SBP and HR during the session (Table 1). At the end of MOD, the RPE was 11 and [Lac] was 3.8 mM, whereas at the end of MAX the RPE reached 19, and [Lac] was 6.7 mM, which shows that MAX was performed at a higher intensity than MOD. According to MacDonald et al.[43], the hemodynamic stress and metabolite accumulations induced by exercise are among the main factors responsible for muscle vasodilatation, and consequently for the decrease in peripheral vascular resistance during and after exercise.

Piepoli et al.[38] conducted three experimental sessions of maximal incremental exercise with 25 W increments every 5 min, moderate exercise with 5 stages at the 12–15 W load, and constant minimal exercise at 50 W of the same duration as the maximal exercise, and a control session with 8 healthy individuals. After exercising, the participants remained for 60 minutes at the facility for BP, peripheral vascular resistance and forearm vascular resistance analysis. Piepoli et al.[38] concluded that, compared to the moderate, minimal and control sessions, only maximal exercise was effective at reducing DBP. This finding was probably due to decreases in peripheral vascular resistance and forearm vascular resistance, which remained lower for 1 h only after maximal exercise. Piepoli et al.[39] also reported that, after ten healthy individuals performed a maximal exercise session, their DBP was reduced for one hour, probably because

of a decrease in peripheral vascular resistance (as cited above), even though there was an increase in renin activity, persistent sympathetic activity and reduced vagal tone, possibly due to peripheral vasodilatation.

Neural and humoral factors also influence BP levels. Reductions in cardiac output and decreases in peripheral vascular resistance are associated with the occurrence of PEH[40, 41]. Two mechanisms have been proposed for explaining the post exercise decrease of peripheral resistance: sympathetic inhibition, and alterations in vascular responsiveness. The activation of the neurokinin-1 receptor during exercise triggers the receptor to undergo internalization after the completion of exercise, dampening the GABA interneuron's solitary tract nucleus, and modifying the baroreflex to a lower level after exercise, by reducing transmission to baroreceptor second-order neurons, increasing the excitement of the ventral caudal lateral medulla, increasing inhibition of the rostral ventral lateral medulla, inhibiting its action, and decreasing sympathetic nervous activity, leading to post-exercise hypotension. On the other hand, a greater local release of nitric oxide, prostaglandins, adenosines, and ATP may also alter the vascular response and contribute to BP reduction[42].

T2D patients can present endothelial dysfunction[2] and reduced release of vasodilator substances, which suggests that a possible mechanism for the decrease of vascular resistance is damaged in this population, as observed in the post-MOD response. Nevertheless, after MAX this limitation would be less evident, as was demonstrated in the present study of T2D patients. Perhaps exercise performed at higher intensities allows greater recruitment of motor units and, consequently, induces greater metabolic and hemodynamic stress, as well as promoting greater BP decrease in the post-exercise period.

The American College of Sports Medicine[9] recommends moderate intensity exercise for individuals with T2D, but higher intensity exercise may have additional benefits for cardiorespiratory fitness and glucose control[44]. Maximal exercise is a very well known and useful method for verifying exercise response. Thus, its use is important for determining the response of special populations such as T2D patients, considering that these individuals tend to present more cardiovascular problems[5]. Nevertheless, it is important to point out that the use of high intensity exercise with T2D patients should be well supervised, and performed only by individuals whose BP and T2D are controlled and who have no associated complications such as cardiovascular dysfunction, and should be used only after having proven the patient's physical and cardiorespiratory fitness in clinical examinations.

Finally, we conclude that a single aerobic exercise session resulted in BP reduction for 24 h in individuals with T2D, particularly while sleeping, and the magnitude of this reduction seems to be dependent on the intensity at which the exercise is performed.

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REFERENCES

1) Association AD American Diabetes Association: Diagnosis and classification of diabetes mellitus. Diabetes Care, 2010, 33: S62–S69. [Medline] [CrossRef]
2) Ding H, Triggle CR: Endothelial cell dysfunction and the vascular complications associated with type 2 diabetes: assessing the health of the endothelium. Vasc Health Risk Manag, 2005, 1: 55–71. [Medline] [CrossRef]
3) Figueroa A, Baynard T, Fernhall B, et al.: Endurance training improves post-exercise cardiac autonomic modulation in obese women with and without type 2 diabetes. Eur J Appl Physiol, 2007, 100: 437–444. [Medline] [CrossRef]
4) Arauz-Pacheco C, Parrott MA, Raskin P, American Diabetes Association: Treatment of hypertension in adults with diabetes. Diabetes Care, 2003, 26: S80–S82. [Medline] [CrossRef]
5) Van de Veire NR, De Winter O, Gir M, et al.: Fasting blood glucose levels are related to exercise capacity in patients with coronary artery disease. Am Heart J, 2006, 152: 486–492. [Medline] [CrossRef]
6) Wild S, Roglic G, Green A, et al.: Global prevalence of diabetes: estimates for the year 2000 and projections for 2030. Diabetes Care, 2004, 27: 1047–1053. [Medline] [CrossRef]
7) Venables MC, Jeukendrup AE: Physical inactivity and obesity: links with insulin resistance and type 2 diabetes mellitus. Diabetes Metab Res Rev, 2009, 25: S18–S23. [Medline] [CrossRef]
8) Zanuso S, Balducci S, Jimenez A: Physical activity, a key factor to quality of life in type 2 diabetic patients. Diabetes Metab Res Rev, 2009, 25: S24–S28. [Medline] [CrossRef]
9) Colberg SR, Albright AL, Blissmer BJ, et al. American College of Sports Medicine American Diabetes Association: Exercise and type 2 diabetes: American College of Sports Medicine and the American Diabetes Association: joint position statement. Exercise and type 2 diabetes. Med Sci Sports Exerc, 2010, 42: 2282–2303. [Medline] [CrossRef]
10) Tamura T, Kida K, Seki T, et al.: Study of relationship between exercise therapy and diet therapy in type 2 diabetes Mellitus patients. J Phys Ther Sci, 2011, 3: 485–488. [CrossRef]
11) Hiyane WS, Moreira SR, Valle G, et al.: Blood glucose responses of type-2 diabetics during and after exercise performed at intensities above and below anaerobic threshold. Braz J Kineanthropometry Hum Perform, 2008, 10: 8–11.
12) Hordern MD, Marwick TH, Wood P, et al.: Acute response of blood glucose to short-term exercise training in patients with type 2 diabetes. J Sci Med Sport, 2011, 14: 238–242. [Medline] [CrossRef]
13) Morais PK, Campbell CS, Sales MM, et al.: Acute resistance exercise is more effective than aerobic exercise for 24h blood pressure control in type 2 diabetics. Diabetes Metab, 2011, 37: 112–117. [Medline] [CrossRef]
14) Lima LC, Assis GV, Hiyane W, et al.: Hypotensive effects of exercise performed around anaerobic threshold in type 2 diabetic patients. Diabetes Res Clin Pract, 2008, 81: 216–222. [Medline] [CrossRef]
15) Motta DF, Lima LC, Arsa G, 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: 363–368. [Medline] [CrossRef]
16) Morton RD, West DJ, Stephens JW, et al.: Heart rate prescribed walking training improves cardiorespiratory fitness but not glycaemic control in people with type 2 diabetes. J Sports Sci, 2010, 28: 93–99. [Medline] [CrossRef]
17) Tamura T, Kida K, Suetsuna F, et al.: Follow-up study of continuation versus discontinuation of home exercise in type 2 diabetes patients. J Phys Ther Sci, 2011, 23: 91–96. [CrossRef]
18) Kenney MJ, Seals DR: Postexercise hypotension. Key features, mechanisms, and clinical significance. Hypertension, 1993, 22: 653–664. [Medline] [CrossRef]
19) Moraes MR, Bacurau RF, Ramalho JD, et al.: Increase in kinins on post-exercise hypotension in normotensive and hypertensive volunteers. Biol Chem, 2007, 388: 533–540. [Medline] [CrossRef]
20) Gomes Anunciação P, Doederlein Polito M: A review on post-exercise hypotension in hypertensive individuals. Arq Bras Cardiol, 2011, 96: e100–e109. [Medline]
21) Quinn TJ: Twenty-four hour, ambulatory blood pressure responses following acute exercise: impact of exercise intensity. J Hum Hypertens, 2000,

14: 547–553. [Medline] [CrossRef]
22) Pescatello LS, Fargo AE, Leach CN Jr, et al.: Short-term effect of dynamic exercise on arterial blood pressure. Circulation, 1991, 83: 1557–1561. [Medline] [CrossRef]
23) Ciolac EG, Guimarães GV, D'Avila VM, et al.: Acute aerobic exercise reduces 24-h ambulatory blood pressure levels in long-term-treated hypertensive patients. Clinics (Sao Paulo), 2008, 63: 753–758. [Medline] [CrossRef]
24) Eicher JD, Maresh CM, Tsongalis GJ, et al.: The additive blood pressure lowering effects of exercise intensity on post-exercise hypotension. Am Heart J, 2010, 160: 513–520. [Medline] [CrossRef]
25) Simões HG, Campbell CS, Kushnick MR, et al.: Blood glucose threshold and the metabolic responses to incremental exercise tests with and without prior lactic acidosis induction. Eur J Appl Physiol, 2003, 89: 603–611. [Medline] [CrossRef]
26) Simões HG, Hiyane WC, Benford RE, et al.: Lactate threshold prediction by blood glucose and rating of perceived exertion in people with type 2 diabetes. Percept Mot Skills, 2010, 111: 365–378. [Medline] [CrossRef]
27) Moreira SR, Simões GC, Hiyane WC, et al.: Identification of the anaerobic threshold in sedentary and physically active individuals with type 2 diabetes. Braz J Phys Ther, 2007, 11: 289–296. [CrossRef]
28) Alessi A, Brandão AA, Pierin A, et al. Sociedade Brasileira de Cardiologia Sociedade Brasileira de Hipertensão Sociedade Brasileira de Nefrologia: [IV Guideline for ambulatory blood pressure monitoring. II Guideline for home blood pressure monitoring. IV ABPM/II HBPM]. Arq Bras Cardiol, 2005, 85: 1–18. [Medline]
29) Katoh J, Hirayama M, Murakami M, et al.: Physical fitness and exercise endurance measured by oxygen uptake kinetics in patients with type 2 diabetes mellitus. J Phys Ther Sci, 2001, 13: 83–85. [CrossRef]
30) Borg GA: Psychophysical bases of perceived exertion. Med Sci Sports Exerc, 1982, 14: 377–381. [Medline] [CrossRef]
31) Svedahl K, MacIntosh BR: Anaerobic threshold: the concept and methods of measurement. Can J Appl Physiol, 2003, 28: 299–323. [Medline] [CrossRef]
32) Tsumiyama W, Oki S, Umei N, et al.: Evaluation of the lactate threshold during downhill running in rats. J Phys Ther Sci, 2014, 26: 125–126. [Medline] [CrossRef]
33) Chobanian AV, Bakris GL, Black HR, et al. Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. National Heart, Lung, and Blood Institute National High Blood Pressure Education Program Coordinating Committee: Seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. Hypertension, 2003, 42: 1206–1252. [Medline] [CrossRef]
34) Kushner RF, Blatner DJ: Risk assessment of the overweight and obese patient. J Am Diet Assoc, 2005, 105: S53–S62. [Medline] [CrossRef]
35) Leite SA, Monk AM, Upham PA, et al.: Low cardiorespiratory fitness in people at risk for type 2 diabetes: early marker for insulin resistance. Diabetol Metab Syndr, 2009, 1: 8. [Medline] [CrossRef]
36) Scott JA, Coombes JS, Prins JB, et al.: Patients with type 2 diabetes have exaggerated brachial and central exercise blood pressure: relation to left ventricular relative wall thickness. Am J Hypertens, 2008, 21: 715–721. [Medline] [CrossRef]
37) Jones H, George K, Edwards B, et al.: Exercise intensity and blood pressure during sleep. Int J Sports Med, 2009, 30: 94–99. [Medline] [CrossRef]
38) Piepoli M, Isea JE, Pannarale G, et al.: Load dependence of changes in forearm and peripheral vascular resistance after acute leg exercise in man. J Physiol, 1994, 478: 357–362. [Medline]
39) Piepoli M, Coats AJ, Adamopoulos S, et al.: Persistent peripheral vasodilation and sympathetic activity in hypotension after maximal exercise. J Appl Physiol 1985, 1993, 75: 1807–1814. [Medline]
40) Rezk CC, Marrache RC, Tinucci T, et al.: Post-resistance exercise hypotension, hemodynamics, and heart rate variability: influence of exercise intensity. Eur J Appl Physiol, 2006, 98: 105–112. [Medline] [CrossRef]
41) Halliwill JR: Mechanisms and clinical implications of post-exercise hypotension in humans. Exerc Sport Sci Rev, 2001, 29: 65–70. [Medline] [CrossRef]
42) Chen CY, Bonham AC: Postexercise hypotension: central mechanisms. Exerc Sport Sci Rev, 2010, 38: 122–127. [Medline] [CrossRef]
43) MacDonald JR, MacDougall JD, Hogben CD: The effects of exercise duration on post-exercise hypotension. J Hum Hypertens, 2000, 14: 125–129. [Medline] [CrossRef]
44) Boulé NG, Kenny GP, Haddad E, et al.: Meta-analysis of the effect of structured exercise training on cardiorespiratory fitness in Type 2 diabetes mellitus. Diabetologia, 2003, 46: 1071–1081. [Medline] [CrossRef]