Involvement of rest diastolic arterial pressure in autonomic heart rate recovery from exercise in normotensive men

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OBJECTIVE: Rest arterial pressure has been shown to be associated with cardiovascular mortality. Autonomic heart rate control during recovery from exercise is estimated to detect changes in cardiovascular system, which may lead to cardiovascular diseases. We assessed the involvement of rest diastolic arterial pressure (DAP) on heart rate dynamics after exercise in normotensive physically active men.
METHOD: We evaluated healthy physically active men aged 18 to 22 years old divided into two unequal groups: G1- rest DAP between 80 and 90 mmHg (N=11) and G2- rest DAP < 80mmHg (N=24). Volunteers performed physical exercise on a treadmill with intensity equivalent to 60% of V_{max}. Heart rate recovery in the first (HRR1) and third (HRR3) minute after exercise were measured and heart rate variability (HRV) was examined in the time and frequency domain. Additionally, we performed the quantitative analysis of the Poincaré plot. HRV was recorded in the following phases: the 10-minute period before exercise, during exercise and the 60 minute period after exercise.
RESULTS: We found no significant difference between G1 and G2 concerning HRV changes during exercise. The G2 group exhibited a delayed recovery of SDNN, RMSSD, RRTri, LF, HF, LF/HF, SD1 and SD2 indices during recovery from exercise. HRR1 and HRR3 was greater in the G2 group.
CONCLUSION: Normotensive physically active men with DAP between 80 and 90 mmHg presented faster heart rate recovery and an accelerated recovery of heart rate autonomic control after aerobic exercise.

KEYWORDS: Arterial Pressure; Autonomic Nervous System; Cardiovascular Physiological Phenomena; Cardiovascular system.

■ INTRODUCTION

Recovery of cardiovascular parameters after aerobic exercise provides suitable information regarding coronary heart disease and incidents of cardiovascular disorders.[1] Also, cardiovascular variables evaluated after exercise provide evidence that may not be recognized at rest.[2]

Heart rate recovery (HRR) following exercise and heart rate variability (HRV) are well recognized methods used to investigate heart rate autonomic regulation.[2,3]

Several variables influence HRR and HRV, including age, level of physical activity and hypertension.[2] Best et al[4] have reported that increased arterial pressure impairs HRR and resting HRV.

In these situations, arterial pressure has been recognized as a strong, independent and direct factor connected with cardiovascular mortality. Lewington et al[5] performed a meta-analysis including one million adults in 61 prospective observational studies and reinforced an association with risk throughout the normal physiological range of systolic (SAP) and diastolic arterial pressure (DAP) (approximately 115/75 mmHg).

The association between arterial pressure and cardiovascular risk doubles with each increase of 20/10 mmHg starting at 115/75 mmHg.[5] Based on this result, the Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC-7) attempted to establish a new range of arterial pressure between normal and pre-hypertension necessary to draw attention to preventive interventions.[6]

It is well known that impaired heart rate autonomic control during recovery from exercise is associated with the prediction of mortality in patients with heart disorders[7] and also in healthy individuals.[8] However, it is unclear whether rest arterial pressure influences cardiovascular parameters during and after exercise in physically active healthy subjects. Furthermore, the investigation of alterations during recovery from exercise in this specific cohort is important to establish innovative values of arterial pressure for cardiovascular risk. Consequently, we investigated the involvement of rest DAP in the responses of heart rate dynamics to aerobic exercise in normotensive men.

■ MATERIALS AND METHODS

Population

This study population exclusively comprised male students. They were all non-smokers, aged between 18 and 22 years; they were divided in two unequal groups: G1: diastolic arterial pressure (DAP) between 80 and 90 mmHg (N=11); G2: DAP < 80mmHg (N=24). All volunteers routinely performed moderate to intense physical activity lasting at least 1 hour for 3 days per week. However, they were not classified as athletes.[9] All volunteers were informed about the procedures of the study and signed a confidential written informed consent. The study adhered to Resolution 466/2012 of the National Health Agency 10/10/1996 and was approved by the Ethics Committee in Research of UNESP (No. CEP-2011-385).

We did not include volunteers: (a) with a body mass index (BMI) > 30 kg/m^2; (b) with Systolic Arterial Pressure (SAP) > 130 mmHg (at rest); (c) with cardiovascular, respiratory and reported neurological disorders; (d) under medication with influences upon the autonomic nervous system.

Cardiovascular variables

Systolic and diastolic arterial pressures were recorded using an aneroid sphygmomanometer (WelchAllyn, Germany) and stethoscope (Littmann, USA); heart rate was obtained through a heart rate monitor - Polar RS800CX (Polar Electro, Kempele, Finland).[10]

Heart rate recovery was calculated as the difference between the heart rate at the termination of exercise and heart rate at 1 min (HRR1) and 3 min (HRR3) after cessation of the protocol.[2]

HRV analysis

We followed the directives laid down by the Task Force guidelines.[3] HRV recording was performed with a transmitter placed on the patients' chest and a heart rate monitor (Polar® RS800CX; Polar Electro Oy, Kempele, Finland), which has been previously validated.[11] Technical details concerning HRV analysis were described in previous studies using identical methodology.[12,13]

Definition of HRV indices included time and frequency domain: Time domain - pNN50: percentage of adjacent RR intervals with a difference of duration greater than 50 milliseconds; SDNN: average standard deviation of normal RR intervals and RMSSD: square root of the average square differences between normal adjacent RR intervals. Geometric domain - RRTri: Triangular index, TINN: Triangular interpolation of RR intervals; SD1: standard deviation of the instantaneous variability of the beat-to beat heart rate; SD2: standard deviation of long-term continuous RR interval variability. Frequency domain - low frequency - LF: 0.04 to 0.15 Hz - and high frequency - HF: 0.15 to 0.40 Hz - in absolute (ms^2) and in normalized units via Fast Fourier Transform.[14] HRV were evaluated before, during and after exercise. When investigating linear indices in the frequency and time domains we used the Kubios HRV® analysis software.[15]

Maximal effort test

We applied 60% of V_{max} determined in the progressive test described below using the concept developed by Conconi et al,[16] which was suggested to assess the anaerobic threshold for detecting the heart rate deflection point utilizing a progressive test through the D_{max} method:[16] this method is used for the identification of the heart rate deflection point, the corresponding heart rate and speed points were plotted. The heart rate values were adjusted by means of a third-degree polynomial function and a linear equation of the first degree, which are data derived from each individual. Next, the difference of the values of heart rate obtained through the above-mentioned equations were calculated and a curve was plotted using these values. We considered the heart rate deflection point as the highest value before a change of direction in curve.[19]

The volunteers underwent an exhaustive progressive treadmill test (Inbrasport ATL 2000) with an initial speed of 8 km/hour, incremented by 1 km/hour at 2 min intervals until volitional exhaustion or onset of clinical changes that prevented the continuity of test, such as dizziness, shortness of breath or intense "air hunger".[17,18] The inclination of the treadmill remained fixed at 1%, since this condition reflects more precisely the energy cost of running outdoors. We only included volunteers that reached up to 90% of maximal heart rate.[19]

Experimental Protocol

Exercise protocols were performed in the same soundproofed room for all subjects. The relative humidity was controlled between 50% and 60% and temperature was maintained between 21°C and 25°C. Volunteers were instructed not to ingest alcohol, caffeine or other autonomic nervous system stimulants for 24 hours before the evaluation. Datasets were collected on an individual basis, always between hours 18:00 and 21:00 to standardize circadian influences.

Volunteers warmed up before the test proper through a treadmill exercise at a speed of 6 km/hour + 1% slope for five minutes. This was followed by 25 minutes at an intensity equivalent to 60% of V_{max} according to the Conconi threshold, at the same 1% slope.

HRV was examined in the following stages: M1 (control): the 10-minute period before the performance of the exercise; during exercise: M2, during the 10 to 15-minute interval of exercise; M3, during the 25 to 30-minute interval of exercise; during recovery from exercise: M4, at 5-10 minutes; M5, at 15-20 minutes; M6, at 25-30 minutes; M7, at 35-40 minutes; M8, at 45-50 minutes; M9, at 55-60 minutes after the end of exercise.

Statistical Analysis

Shapiro-Wilk goodness-of-fit test evaluated the normality of distributoins (z value > 1.0)[20].

We applied the Mann-Whitney test for non-parametric distributions and the non-paired Student t-test for parametric distributions in order to compare variables between groups.

To evaluate variables in the exercise protocol (control at rest, during exercise and post-exercise) we used a two-way repeated measures analysis of variance test followed by the Bonferroni post-hoc test for parametric distributions and a Friedman test followed by Dunn's test for non-parametric distributions.

We considered differences to be significant when $p < 0.05$ (5%)

■ RESULTS

Table 1 shows descriptive statistics regarding weight, height, BMI, DAP, SAP, HRR1 and HRR3. Comparing Groups 1 vs. 2, we observed significantly higher values for BMI, systolic and diastolic arterial pressure in G1 while heart rate recoveries at 1 and 3 minutes were more intense in G2.
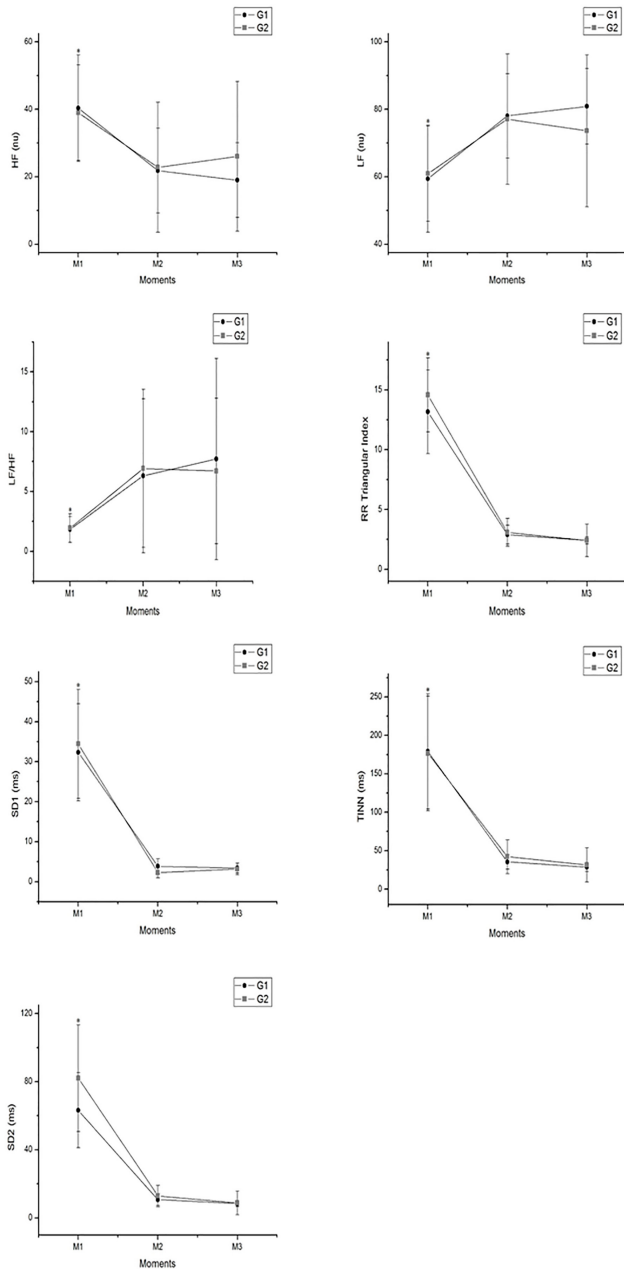
Figures 1 shows geometric and frequency domain indices before and during exercise. At 15 and 30 minutes of exercise we noted reduced values of HF, RR triangular index, TINN, SD1 and SD2 compared to rest before exercise in both groups. However, we noted no significant difference between groups.

Figure 2 displays time domain indices of HRV before and during exercise. All indices were reduced at 15 and 30 minutes of exercise. Nonetheless, there was no difference between groups.

Table 1 - Descriptive statistics of weight, height, body mass index (BMI), diastolic (DAP) and systolic arterial pressure (SAP) and heart rate recovery at 1 min (HRR1) and 3 min (HRR3) after termination of the exercise of the volunteers divided by group.

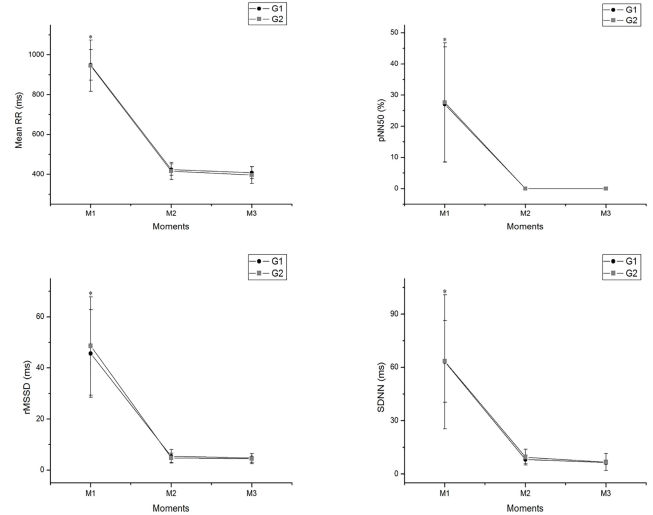| G1	MEAN ± STD DEV.	MEDIAN	MINIMUM	MAXIMUM	P (G1 VS. G2)
AGE (YEARS)	21.6 ± 1.6	22	19	24	0.42
HEIGHT (M)	1.75 ± 0.05	1.75	1.65	1.84	0.22
WEIGHT (KG)	78.6 ± 8,7	79.4	65.7	93.9	0.11
BMI (KG/M2)	25.73 ± 1.43	26.1	22.32	28.8	0.011
SAP (MMHG)	120.0 ± 5.8	120	110	130	<0.001
DAP (MMHG)	80.8 ± 2.8	80	80	90	<0.001
HRR1 (BPM)	15.9 ± 7.9	14	4	33	0.02
HRR3 (BPM)	33.4 ± 9.8	29	21	50	0.03
G2					
AGE (YEARS)	21.8 ± 3.1	21	18	29	
HEIGHT (M)	1.77 ± 0.01	1.79	1.6	1.88	
WEIGHT (KG)	74.4 ± 10.9	73.1	57	97	
BMI (KG/M2)	23.7 ± 2.3	23.63	19.33	28.65	
SAP (MMHG)	105.0 ± 10.6	105	90	130	
DAP (MMHG)	64.5 ± 5.1	60	60	70	
HRR1 (BPM)	24.7 ± 13.5	18.5	2	57	
HRR3 (BPM)	42.6 ± 15.3	40	24	87	

m: meters; kg: kilograms; p indicates difference between G1 VS G2.

Figure 2: Time domain analysis of HRV before and during exercise. M1: Control at rest; M2: 10-15 minutes after start of exercise; M3: 25-30 minutes after start of exercise; G1: Group with SAP > 120 mmHg; G2: Group with SAP < 120 mmHg; pNN50: the percentage of adjacent RR intervals with a difference of duration greater than 50 ms; RMSSD: root-mean square of differences between adjacent normal RR intervals in a time interval; ms: milliseconds; SDNN: Average standard deviation of normal RR intervals; * p<0.05 Vs. M2 and M3.

Figure 1: Frequency domain and geometric analysis of HRV before and during exercise. M1: Control at rest; M2: 10-15 minutes after start of exercise; M3: 25-30 minutes after start of exercise; G1: Group with DAP ± 80 mmHg; G2: Group with DAP < 80 mmHg; LF: low frequency; HF: high frequency; LF/HF: low frequency/high frequency ratio; ms: milliseconds; SD1: standard deviation of the instantaneous variability of the beat-to-beat heart rate; SD2: standard deviation of long-term continuous RR interval variability; TINN: Triangular interpolation of RR interval histogram. *p<0.05 Vs. M2 and M3.

Figure 3 presents HRV indices in frequency and geometric domains during recovery from exercise. As expected, HRV was reduced during recovery from exercise compared to rest before exercise except TINN and LF/HF.

In Group 1 the LF and LF / HF ratio were increased at M4 compared to M1. In Group 2 the LF, HF, and LF / HF ratio was statistically different at M1 compared to M4, M5 and M7 (Figure 3).

TINN was not statistically different between time periods in both groups (Figure 3).

Figure 3 also illustrates SD1 and SD2 indices in both groups, at rest and during recovery from exercise. Both indices decreased during recovery from exercise. In G1 the SD1 index was statistically decreased at M4 and M5 compared to M1, although the SD2 index was also reduced at M4 and M5 compared to M1. Furthermore, in G2 the SD1 index was significantly weakened at M4, M5 and M6 compared to M1 while in G1 the SD2 index was significantly decreased in M4 compared to M1 (Figure 3).
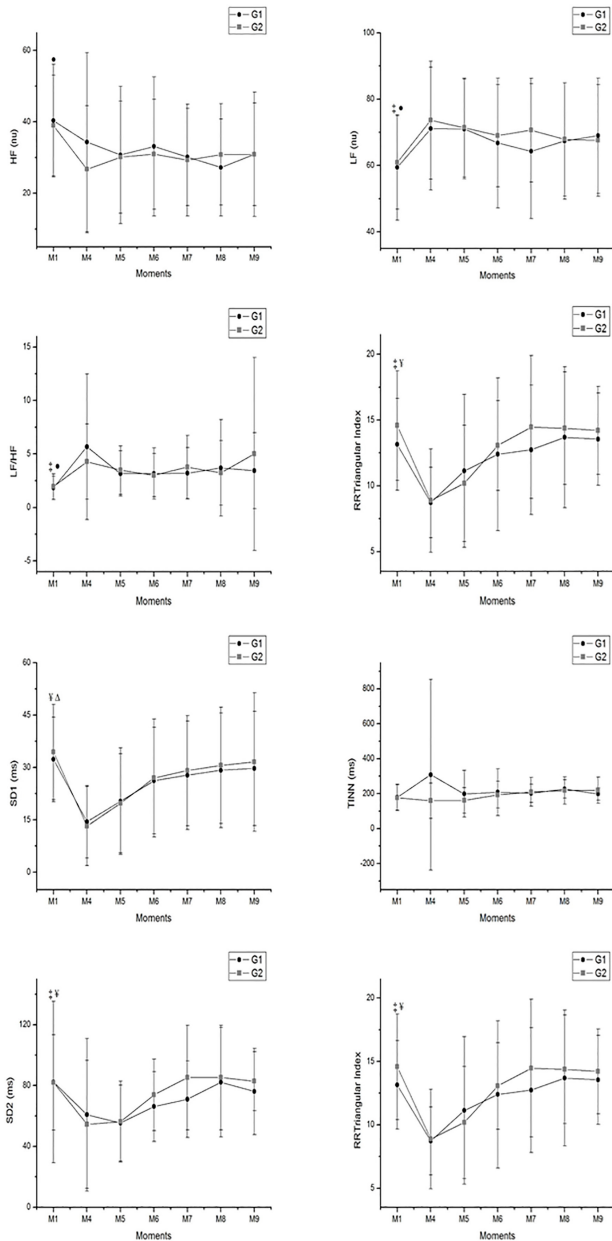
Figure 4 presents HRV indices in the time domain during recovery from exercise. We reported significant changes in all indices during recovery from exercise compared to before exercise in both groups.

The mean RR interval in G1 at rest (M1) was statistically diminished from all other times, while in G2 it was lessened compared to the M4 , M5, M6 and M7. Regarding the pNN50 index we observed a significant difference in relation to M1, M4 and M5 in both groups, as well as for rMSSD index in G1 and SDNN and RR Tri indices in G2. The SDNN and RR Tri indexes were dissimilar between M1 and M4 in G1. In G2 the rMSSD was reduced in M4, M5 and M6 compared to M1 (Figure 4).
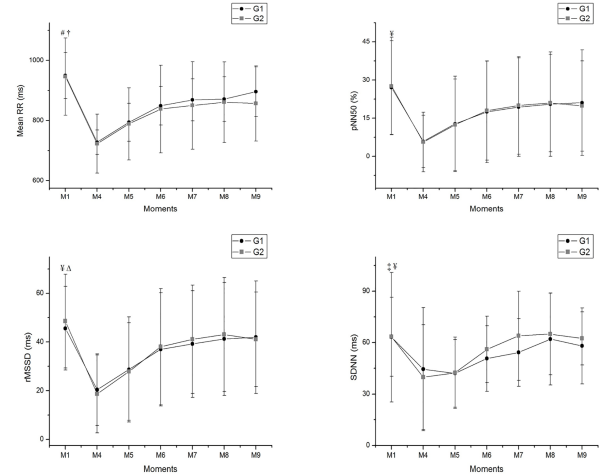
■ DISCUSSION

Abnormal heart rate during recovery from aerobic exercise has been associated with the forecast of the development of cardiovascular disease.[21,22] Here, our

Figure 4: Time domain analysis of HRV before and after exercise. M1: Control at rest; M4: 5-10 minutes after exercise cessation; M5: 15-20 minutes after exercise cessation; M6: 25-30 minutes after exercise cessation; M7: 35-40 minutes after exercise cessation; M8: 45-50 minutes after exercise cessation; M9: 55-60 minutes after exercise cessation; G1: Group with DAP > 80 mmHg; G2: Group with DAP < 80 mmHg. #p<0.05 Vs. M4, M5, M6, M7, M8 and M9; † p<0.05 Vs. M4, M5, M6 and M7; ¥ p<0.05 Vs. M4 and M5; Δ p<0.05 Vs. M4, M5 and M6.

Figure 3: Frequency domain and geometric analysis of HRV before and after exercise. M1: Control at rest; M4: 5-10 minutes after exercise cessation; M5: 15-20 minutes after exercise cessation; M6: 25-30 minutes after exercise cessation; M7: 35-40 minutes after exercise cessation; M8: 45-50 minutes after exercise cessation; M9: 55-60 minutes after exercise cessation; G1: Group with DAP > 80 mmHg; G2: Group with DAP < 80 mmHg; LF: low frequency; HF: high frequency; LF/HF: low frequency/ high frequency ratio; ms: milliseconds; SD1: standard deviation of the instantaneous variability of the beat-to-beat heart rate; SD2: standard deviation of long-term continuous RR interval variability; TINN: Triangular interpolation of RR interval histogram. ‡ p<0.05 Vs. M4; ¥ p<0.05 Vs. M4 and M5; Δp<0.05 Vs. M4, M5 and M6; • p<0.05 Vs. M4, M5 and M7.

investigation intended to evaluate the involvement of diastolic arterial pressure in autonomic heart rate responses to aerobic exercise and during recovery in physically active normotensive men. We noticed the following consequences: 1) Heart rate parasympathetic reactivation after aerobic exercise was more accelerated in subjects with DAP between

80 and 90 mmHg; 2) The sympatho-vagal balance decline during recovery from exercise was also more accelerated in volunteers with DAP between 80 and 90 mmHg; 3) HRR was faster in men with DAP between 80 and 90 mmHg and; 4) No difference was observed during exercise between groups.

Increases in arterial pressure and heart rate during physical exercise are shown to distinguish risk factors related to the development of hypertension. This is meticulously described in a earlier study which reported that more intense increase in arterial pressure and heart rate responses induced by exercise was associated with prediction of hypertension.[23]

Also, reliable signs indicate that hypertensive patients have autonomic nervous system dysfunction characterized by reduced parasympathetic tone and higher sympathetic activity analyzed through pharmacological parasympathetic and sympathetic blockade, salivary flow and HRV monitoring.[24-26]

In this way, we had predicted that healthy physically active men with higher levels of diastolic arterial pressure would exhibit a delayed heart rate autonomic recovery following exercise. Remarkably, late recovery of heart rate autonomic control after exercise was found in males with DAP < 80mmHg compared to normotensive subjects with DAP between 80 and 90 mmHg. We found that RMSSD, SD1 and the HF band recovered more slowly from exercise in the group with lower Diastolic Pressure.

We also documented that sympatho-vagal recovery from exercise evaluated through HRV was delayed in men with lower Diastolic Pressure, strengthening the involvement of resting Systolic and Diastolic Pressure on autonomic heart rate control after physical exercise.

Our conclusions contradict Shin *et al*,[27] who investigated pre-hypertensive subjects: they included pre-hypertensive volunteers with SAP between 120 and 139 mmHg or DAP between 80 and 89 mmHg. The pre-hypertensive group displayed decreased heart rate recovery after exercise compared to a control group comprising normotensive subjects, which would indicate that heart rate autonomic dysfunction is present before development of hypertension. Our results show that HRR was slower in normotensive men with lower diastolic pressure compared to normotensive men with higher diastolic pressure. Equally, a recent study reported an association between autonomic heart rate function during recovery from exercise and development of hypertension.[28] Jae et al evaluated HRR of 1855 normotensive men with risk of incident hypertension. After a four year follow-up it was concluded that slow HRR was associated with hypertension development in normotensive men.

In this context, it has been previously reported that pre-hypertensive subjects already present delayed HRR.[29] Yet, there are important differences between the Erdogan et al study[29] and our investigation regarding methodological issues. The authors considered pre-hypertension as SAP between 120 and 139 mmHg and/or DAP between 80 and 89 mmHg. The pre-hypertensive patients evaluated were older (48 ± 8 years old), had higher BMI (29.1 ± 4 kg/m^2) and included males and females. Our study only selected healthy physically active males between 18 and 22 years old.

The delayed HRV recovery and slower HRR after exercise in males with DAP < 80 mmHg could be explained by the influence of metaboreflex on baroreflex. During exercise muscle afferents sensitive to metabolites (K$^+$, H$^+$, ADP and Pi for instance) activate the metaboreflex, which resets baroreflex function through brainstem levels and decreases its sensitivity.[30,31] Immediately after exercise parasympathetic reactivation and sympathetic withdrawal regulate HRR and HRV during the recovery phase.[32] A well acknowledged theory is that the removal of metabolites from the muscles after exercise would progressively reduce metaboreceptor activity and gradually restore barocptor function. This would then induce a decline in sympathetic and an increase in parasympathetic ctivity to the heart, resulting in HRR.[2]

Accordingly, we hypothesize that in physically healthy men with lower DAP the slower blood flow could result in a late reactivation of baroreflex, leading to slower HRR and delayed HRV recovery from exercise. However, we did not measure blood flow, plasma metabolites and lactate levels to confirm this supposition.

However, our study does highlight some important points which need to be addressed. SAP was higher in the group with higher DAP. This led us to conclude that not only DAP was involved in delayed HRV recovery. BMI was also higher in the group with higher DAP; considering the well established influence of BMI on cardiovascular responses to exercise, which indicates that overweight subjects have impaired autonomic recovery.[33,34] Nevertheless, in this study the group with lower BMI (DAP < 80mmHg) presented delayed heart rate autonomic recovery and slower HRR.

A lmitation of our studies is the unsimilar sizes of the two groups. Similar sample sizes between groups is advisable in a future study.

■ CONCLUSION

Normotensive healthy young men with a DAP between 80 and 90 mmHg presented faster recovery of heart rate autonomic control and more accelerated heart rate recovery after aerobic exercise, when compared to men with DAP lower than 80 mm Hg.

■ ACKNOWLEDGEMENTS

■ CONFLICT OF INTEREST

The authors declare no conflict of interests regarding this study.

■ AUTHOR CONTRIBUTION

All authors participated in the acquisition of data and revision of the manuscript, RLG, LCMV and RDR performed statistical analysis and draft the manuscript. FMV, DMG, LCA and VEV determined the design, interpreted the data and drafted the manuscript. All authors read and gave final approval for the version submitted for publication.

PAPEL DA PRESSÃO ARTERIAL DIASTÓLICA NA RECUPERAÇÃO DA FREQUÊNCIA CARDÍACA APÓS EXERCÍCIO EM HOMENS NORMOTENSOS

INTRODUÇÃO: A pressão arterial de repouso demonstrou estar associada à mortalidade cardiovascular. O controle autonômico da frequência cardíaca durante a recuperação pós-exercício é estimado para detetar mudanças no sistema cardiovascular, porque tais mudanças podem levar a doenças cardiovasculares. Avaliamos o envolvimento da pressão arterial diastólica de repouso (PAD) na dinâmica da frequência cardíaca após o exercício em homens normalmente ativos fisicamente normotensos.

MÉTODO: Avaliamos homens saudáveis e fisicamente ativos com idades compreendidas entre os 18 e os 22 anos divididos em dois grupos desiguais: PAD de relaxamento G1

entre 80 e 90 mmHg (n=11) e PAD <80mmHg (n=24). Os voluntários realizaram exercícios físicos em uma esteira com intensidade equivalente a 60% da Vmax. A recuperação da frequência cardíaca no primeiro (HRR1) e terceiro (HRR3) minuto após o exercício foi medida e a variação da frequência cardíaca (VFC) foi examinada no domínio do tempo e da frequência. Além disso, realizamos a análise quantitativa da trama de Poincaré. A VFC foi registrada nas seguintes fases: os períodos de 10 minutos antes do exercício, durante o exercício e os períodos de 60 minutos após o exercício.

RESULTADOS: Não encontramos diferença significativa entre G1 e G2 em relação às alterações da VFC durante o exercício. O G2 apresentou uma recuperação tardia dos índices SDNN, RMSSD, RRTri, LF, HF, LF/HF, SD1 e SD2 durante a recuperação do exercício. HRR1 e HRR3 foi maior no G2.

CONCLUSÃO: Homens normais e fisicamente ativos com PAD entre 80 e 90 mmHg apresentaram RHE mais rápida e recuperação mais acelerada do controle autonômico da freqüência cardíaca após o exercício aeróbio.

PALAVRAS-CHAVE: Pressão arterial; Sistema nervoso autônomo; Fenômenos Fisiológicos Cardiovasculares; Sistema cardiovascular.

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