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Post-exercise blood pressure responses and cardiac stress after inspiratory muscle training in COPD patients: a pilot study

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KEYWORDS

Respiratory muscle training;
Rate pressure product;
Heart rate variability;
Cardiovascular response;
Blood pressure.

ABSTRACT

Acute responses following a single bout of exercise have been shown to predict blood pressure regulation and cardiovascular adaptations. Inspiratory muscle exercise (IMT) has been utilised in pulmonary rehabilitation programmes for COPD patients, but little is known about the immediate effects of IMT exercise on post-exercise blood pressure and cardiac responses. The current study aimed to investigate acute post-exercise blood pressure, cardiac autonomic, and myocardial oxygen demand responses to a single session of IMT among COPD individuals. Ten male COPD patients, with an average age of 65.44 ± 4.38 years, volunteered for the study. Subjects underwent the IMT protocol at 60% maximal inspiratory pressure as an inspiratory load (6 breaths/set, 1-minute rest between sets, 5 sets), while the control group subjects performed breathing without inspiratory load. Blood pressure and heart rate variability were measured before and immediately, 5, 15 and 30 min after the exercise. Systolic blood pressure in the IMT group was significantly higher than in the control group immediately after exercise. Sympathetic cardiac autonomic modulation and sympathovagal balance also showed similar responses to systolic blood pressure. The rate pressure product index of myocardial oxygen demand and heart rate significantly increased from baseline compared to immediate post-IMT exercise. However, all variables returned to the baseline values within 15 min following exercise, and no adverse effects were reported after the IMT programme. Our data suggest that single bouts of IMT sessions neither elicit post-exercise hypotension nor a high cardiac autonomic response in patients with COPD. Thus, moderate-intensity IMT exercise with a short duration appears to be safe for this population.

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Introduction

Chronic obstructive pulmonary disease (COPD) is a common chronic respiratory disease that is the leading cause of mortality worldwide[1]. COPD patients commonly present with respiratory muscle dysfunction, leading to physical activity intolerance[2].

Inspiratory muscle training (IMT), a series of resistive breathing exercises that aim to promote respiratory muscle strength, is considered part of a pulmonary rehabilitation programme. Previous evidence has confirmed the efficacy of IMT on strengthened respiratory muscle, attenuated dyspnoea and improved exercise performance in patients with COPD[3-5]. Apart from the beneficial effect on the pulmonary system, the blood pressure lowering effect of IMT has also been investigated according to autonomic cardiovascular modulations following the implementation of IMT exercise[6]. A recent meta-analysis demonstrated that long-term IMT intervention decreased systolic blood pressure and diastolic blood pressure in healthy individuals and hypertensive patients and was in part mediated by an alteration in cardiac autonomic activity[6]. In addition, it is suggested that the chronic lowering of resting blood pressure following exercise training may be due to a repeated post-exercise hypotension (PEH) response[7,8]. Focus on acute exercise responses, a single bout of IMT exercise with a load of 30% of maximum inspiratory pressure (MIP) for 15 min, induced post-exercise hypotension together with improved heart rate variability (HRV) in young smokers[9]. By contrast, no significant change in blood pressure and cardio-autonomic activity has been reported after acute IMT at 60% maximum inspiratory pressure (PI_{max}) as a resisted load in patients with type 2 diabetes[10]. A recent study showed that the acute effect of IMT at an intensity of 70% MIP had no influence on systolic blood pressure, diastolic blood pressure, HRV or inflammatory markers in obstructive sleep apnoea subjects[11]. As mentioned, the acute effect of IMT exercise on blood pressure response is controversial.

The rate pressure product (RPP) is a marker of myocardial oxygen demand. Moreover, increased RPP also predicts cardiovascular mortality[12-14]. It is well known that cardiac autonomic impairment has been observed in COPD patients and is associated with the development of cardiovascular disease[15,16]. However, little is known about the post-exercise blood pressure, cardiac autonomic responses, and myocardial oxygen demand during a single session of inspiratory muscle exercise in COPD patients. Thus, the present study focused on investigating the acute effect of IMT on post-exercise blood pressure responses, HRV, and rate pressure products in COPD patients. We hypothesised that IMT would alleviate blood pressure and myocardial oxygen demand; these effects may be related to cardiac autonomic modulation.

Materials and methods

Participants

This study recruited 10 pilot COPD patients aged 40–70 years with GOLD stages II–III, following the Global Initiative for Chronic Obstructive Lung Disease guidelines[17]. All patients were clinically stable with absence of respiratory exacerbation within eight weeks prior to the study and had not been involved in any exercise programme. Participants were excluded if they had neurological or musculoskeletal problems that could limit the exercise protocol. We declare that all participants read and signed an informed consent form for study participation, and this study was approved by Khon Kaen University institutional review board (HE622217).

Study design

The participants were randomly divided into either the control or the IMT group using the stratified randomisation method. One week before the measurement, the participants were familiarised with the experimental procedures, and IMT breathing was supervised by a physical therapist[18]. Furthermore, participants were advised to refrain from alcohol, caffeine beverages

and vigorous exercise in the 24 hours preceding testing.

On the test day, the participants were asked to rest for 10 min before the experiment, as described in figure 1. First, baseline measurements were collected: the outcome parameters, including BP, HR, HRV, RPP, and oxygen saturation (SpO_2). Participants in the IMT group received a single session of IMT exercises, while the control group received breathing exercises. After the exercise session, all the participants'variables were measured immediately after the exercise and in the recovery period at 5 min, 15 min, and 30 min after the exercise. Additionally, adverse effect symptoms were recorded for the purpose of investigating the safety of the IMT protocol.

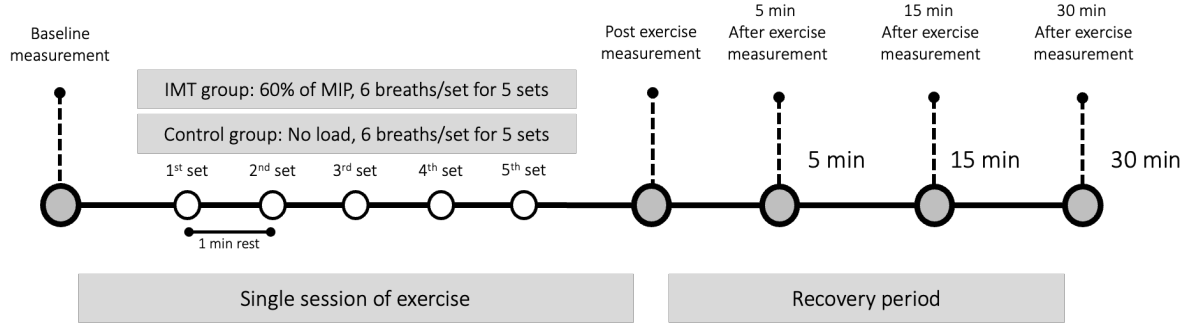

Figure 1 Study protocol and outcome measurement timeline.

Inspiratory muscle training protocol

Prior to the IMT exercise, the MIP was measured for each individual using a MicroRPM respiratory pressure meter (PhysioParts, UK). Participants performed three trials of maximum inspiratory manoeuvre with a 1-minute rest interval between tests. The best of the three trials was used as resistant load, with values that varied no more than 5% of the highest value[19].

The IMT group performed IMT exercises using threshold inspiratory muscle trainer equipment (Koninklijke Philips N.V., Netherlands), and the IMT protocol was set as previously described[9,20]. After attaching the mouthpiece and nose clip, participants were instructed to inhale from the residual volume to overcome the resisted spring load at 60% of their MIP. Participants completed IMT for six breaths per set, for five sets with 1-minute rest between each set. On the other hand, the control group received a single session of breathing exercises with the same protocol, except for no inspiratory load. During the training session, all participants were recommended to maintain diaphragmatic breathing and inhale with maximum effort against load with the same resting breathing frequency. In addition, participants were instructed to stop the exercise if adverse effects occurred, including increasing breathlessness, chest discomfort, dizziness, or near syncope[21].

Outcomes

BP, HR, and SpO_2 were measured using a vital sign monitor (Nihon Kohden Vismo, Japan). The measurement procedure followed the standard guidelines of the American Heart Association[22]. BP was collected three times with at least a 1-minute rest interval. The average value was recorded and used for further analysis.

HRV was measured in a quiet room according to previous guidelines[23]. It was continuously assessed by the R-R interval via electrocardiogram (EKG) using a computer and Acknowledge data collection software (Biopac Systems, USA). The EKG was recorded at 5 min before IMT exercise,

5 min immediately after IMT exercise and at 5–10, 15–20 and 30–35 min after IMT exercise. The time domain (standard deviation of all normal-to-normal R-R intervals [SDNN]) and the frequency domain (high frequency normalised unit [HF], low frequency normalised unit [LF] and low frequency to high frequency ratio [LF/HF]) of the HRV were averaged and analysed for each participant[24]. SDNN reflects all the cyclic components responsible for variability in the period of recording; therefore, it represents total variability. The HF was used as marker of vagal modulation, the LF as index of sympathetic modulation, and the LF/HF represented sympathovagal balance[23].

RPP, a marker of myocardial oxygen demand and cardiac work[25,26], was calculated by multiplying systolic blood pressure (SBP) and HR. During and after completing the exercise, the participants were asked about incidents of adverse effect symptoms[27].

Statistical analysis

Statistical analysis was performed using Statistical Package for Social Sciences (SPSS) version 26 and the Shapiro-Wilk test for normality of distribution was applied. Continuous data were presented as mean ± standard deviation. The data were analysed using a two-way repeated measure ANOVA, followed by Tukey's post hoc test to evaluate specific differences between means. An alpha level of < 0.05 was considered statistically significant.

Results

Participant characteristics

All participants were male, with an average age of 65.44 ± 4.38 years. Baseline characteristic data for each group are summarised in table 1. No statistical differences were observed between the groups in terms of their characteristics.

Table 1 Participant characteristics

	Control group (n=5)	IMT group (n=5)
Age (year)	62.50 ± 2.38	67.80 ± 4.38
Weight (kg)	56.25 ± 4.27	48.98 ± 6.33
Height (cm)	161.1 ± 8.60	160.40 ± 5.03
BMI (kg/m^2)	21.77 ± 2.69	18.98 ± 1.62
MIP (cmH_2O)	59.50 ± 20.04	58.20 ± 10.80

Note: Data present as mean ± SD.

Abbreviation: BMI, Body mass index; MIP, Maximum inspiratory pressure; kg, Kilogram; cm, Centimeter; kg/m^2, Kilogram per square meter; cmH_2O, Centimeter of water.

Blood pressure responses to inspiratory muscle training

Figure 2 shows the SBP and DBP responses before and after IMT. No significant difference in baseline blood pressure was observed between the control and IMT groups. At immediate post-exercise, SBP was significantly elevated following IMT compared with the initial value, while the control group showed a significant reduction in SBP. In addition, SBP was higher after a single bout of IMT than the control condition during immediate post-exercise (p-value < 0.05), and then returned to resting baseline values within 15 min in both groups. In contrast, DBP was not significantly altered at any time point either with the IMT exercise or the control group (Figure 2).

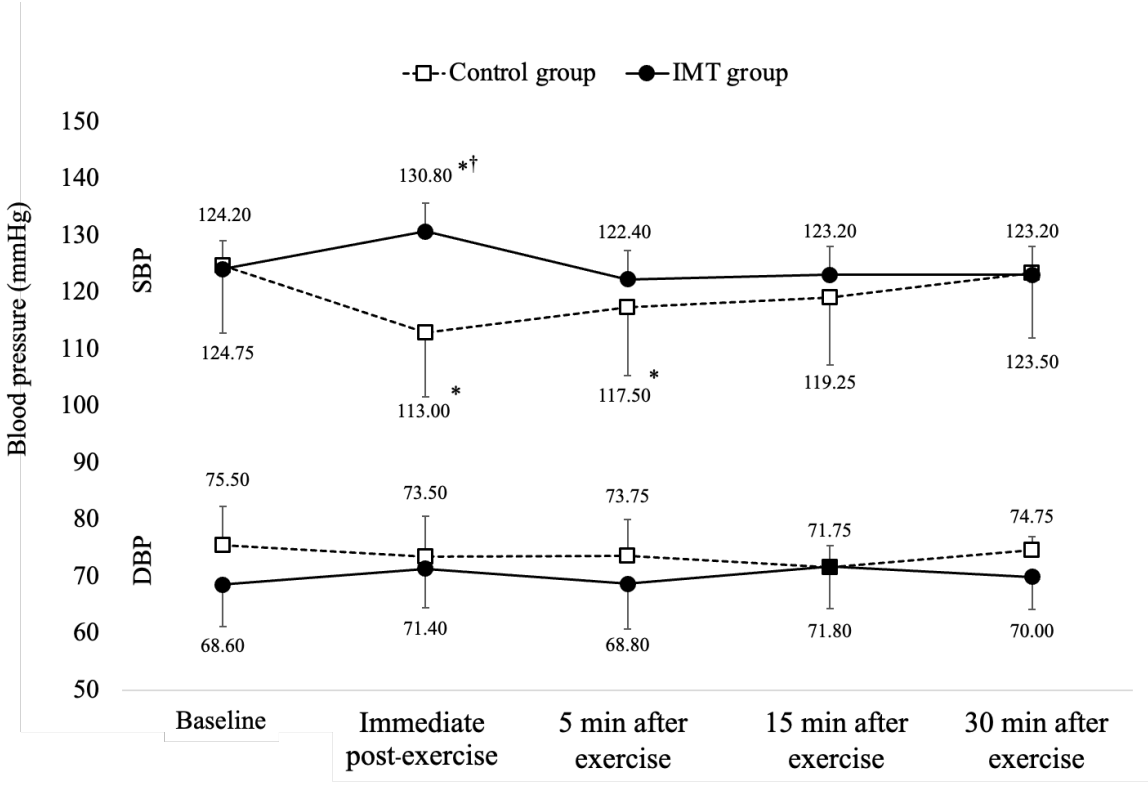

Figure 2 Time course of change in SBP and DBP in the control group and IMT group at baseline, immediate post-exercise, 5 minutes, 15 minutes, and 30 minutes after exercise.

Note: * *p*-value < 0.05 vs. Baseline; † *p*-value < 0.05 vs. the control group.

Abbreviation: SBP, Systolic blood pressure; DBP, Diastolic blood pressure; mmHg, Millimeter of mercury.

Heart rate and rate pressure product responses to inspiratory muscle training

In response to the IMT exercise, HR was increased during the immediate post-exercise compared to baseline values (*p*-value < 0.05, Table 2). However, HR remained unchanged in the control group. The post-hoc test indicated that HR was not significantly different between the conditions. A significant increase in RPP was observed within the IMT group during immediate post-exercise; this was unchanged within the control group. In addition, the inter-group analysis revealed no significant differences between the IMT and the control groups (*p*-value > 0.05). Indeed, HR and RPP returned to resting values during the recovery period after the IMT session (Table 2).

Autonomic responses to inspiratory muscle training

Table 2 presents the HRV responses to the IMT exercise among the COPD patients. The IMT group exhibited a significant within-group reduction in SDNN and HF during immediate post-exercise compared to the rest (*p*-value < 0.05, Table 2). LF and LF/HF were significantly increased responses to a bout of IMT exercise. On the other hand, the control group showed significantly lower LF when compared to the baseline values (*p*-value < 0.05).

The analysis between the groups indicated that IMT exercise resulted in lower HF and higher LF and HF/LF values immediately post-exercise compared to the control group. Accordingly, no significant differences between groups in HRV were observed at 5, 15 and 30 min after exercise.

SpO_2 and adverse effects

There was no significant change in SpO_2 throughout the time period in both groups (*p*-value > 0.05, Table 2). Furthermore, no adverse events were reported in any of the patients during the exercise sessions.

Table 2 Variables in control and inspiratory muscle training (IMT) group at baseline, immediate post-exercise, and recovery period

Variables	Group	Baseline	Immediate post-exercise	5 min after exercise	15 min after exercise	30 min after exercise
HR (bpm)	Control	82.25 ± 15.28	79.25 ± 12.58	81.25 ± 14.59	79.25 ± 16.01	79.25 ± 15.09
	IMT	71.60 ± 6.35	78.00 ± 9.08*	72.60 ± 10.50	69.20 ± 9.36	69.20 ± 7.69
RPP	Control	9865.20 ± 2109.36	9403.60 ± 2061.71	9802.40 ± 2282.50	9648.80 ± 2407.81	9895.20 ± 2338.14
(mmHg.bpm)	IMT	8919.20 ± 1261.10	10238.60 ± 1751.12*	8925.00 ± 1724.51	8557.20 ± 1537.21	8570.40 ± 1611.45
SDNN (ms)	Control	30.15 ± 5.50	31.83 ± 14.18	26.15 ± 8.51	27.98 ± 7.47	26.99 ± 9.81
	IMT	33.42 ± 17.69	23.04 ± 12.15*	28.10 ± 11.16	35.58 ± 19.87	35.47 ± 15.58
HF (n.u.)	Control	64.07 ± 5.04	72.86 ± 3.35	66.32 ± 4.92	62.50 ± 9.21	54.56 ± 14.02
	IMT	58.07 ± 10.11	34.70 ± 21.36*†	52.65 ± 16.20	61.23 ± 10.41	58.89 ± 11.82
LF (n.u.)	Control	35.47 ± 4.88	26.72 ± 4.21*	38.50 ± 14.65	30.87 ± 6.33	30.46 ± 5.86
	IMT	40.96 ± 8.99	66.78 ± 21.92*†	45.52 ± 15.45	37.49 ± 8.35	35.79 ± 10.37
LF/HF	Control	0.56 ± 0.13	0.40 ± 0.14	0.60 ± 0.29	0.51 ± 0.16	0.58 ± 0.14
	IMT	0.75 ± 0.35	2.75 ± 1.81*†	1.05 ± 0.76	0.64 ± 0.26	0.64 ± 0.29
SpO_2 (%)	Control	96.75 ± 0.96	98.00 ± 0.00	96.25 ± 1.50	96.50 ± 1.29	97.00 ± 1.15
	IMT	96.60 ± 2.60	97.00 ± 3.39	96.80 ± 3.42	96.60 ± 3.05	96.80 ± 2.86

Note: Data present as mean ± SD; * significant difference within group when compared with baseline (*p*-value < 0.05); † significant difference between group (*p*-value < 0.05).

Abbreviation: HR, Heart rate; RPP, Rate pressure product; SDNN, Standard deviation of all normal to normal R-R intervals; HF, High frequency normalized unit; LF, Low frequency normalized unit; LF/HF, Low frequency to high frequency ratio; SpO_2, Oxygen saturation.

Discussion

The present study examined changes in post-exercise blood pressure, cardiac autonomic and myocardial oxygen demand in response to one session of inspiratory muscle training in moderate-severe COPD individuals. We demonstrated that no post-exercise hypotension occurred after moderate-intensity IMT exercise among COPD patients. In addition, acute IMT exercise in COPD patients did not provoke cardiac overload as assessed by RPP in comparison with control conditions. Lastly, there were no adverse events, such as increased breathlessness, chest discomfort, dizziness, or near syncope, during the IMT session.

Our results are in agreement with those in a previous study that reported no evidence of post-exercise after completing a single bout of IMT exercise. Findings by Tanriverdi et al[28] exhibited a rise in mean blood pressure (~5%) after performing an acute IMT at an intensity of 60% MIP in healthy young participants. Furthermore, an acute session of inspiratory exercise using a load of 70% MIP in obstructive sleep apnoea subjects did not induce significant changes in blood pressure and heart rate[11]. In contrast, previous studies have demonstrated that breathing exercise at 30% MIP attenuated systolic blood pressure (~13%) and improved cardiac autonomic modulations in young smokers[9]. Thus, in the current study, the

elevation of SBP (~5%) was observed immediately after IMT exercise. A possible explanation relates to the respiratory metaboreflex activation during exercise, which is stimulated by the accumulation of metabolite products from respiratory muscle fatigue[29]. Metaboreflex activation has been consistently reported to increase blood pressure by increasing sympathetic outflow. Previous research has shown that diaphragm muscle fatigue is associated with sympathetic overactivity, leading to increased HR and BP[30]. Indeed, our present study showed that the rise in systolic blood pressure lasted for 5 min following an IMT session using 60% MIP, suggesting that the acute IMT exercise did not induce major haemodynamic changes in moderate-severe COPD participants.

Cardiac autonomic imbalance is considered a potential predictor of cardiovascular events and has also been observed in individuals with COPD. A previous study reported a reduction in HVR among COPD patients, including low vagal modulation, with a predominance of sympathetic activity[31,32]. IMT has been described as an exercise therapy that may improve heart autonomic control according to the interactions of breathing patterns and HR regulation[33]. In older adults, short-term exercise breathing (15 breaths at a load of 30% MIP) induced beneficial changes in the HRV component, as indicated by enhanced LF and attenuated HF[34]. In contrast, the findings of this study revealed that in COPD individuals, a single bout of IMT session with 60% MIP increases sympathetic and decreases parasympathetic activity at 5-minute post-exercise and returns to baseline values at 15-minute post-exercise. Similar to our results, Schein et al. also showed a decrease in vagal modulation following an inspiratory exercise with 60% of PI_{max} in subjects with type 2 diabetes[10]. These findings may be explained by the influence of loading intensity on cardiac autonomic modulation. Prior studies reported that low-intensity IMT produced a greater parasympathetic modulation than high-intensity IMT[24,28]. Nevertheless, the present study showed that the HRV indices were not different in responses to IMT compared to the control group during 5–30 min post-exercise. A previous study also found that HF and LF components were unchanged during acute resistive load breathing using 70% MIP in moderate to severe OSA individuals, suggesting the clinical safety of implementing IMT in this population[11]. These contradictions in blood pressure and cardiac autonomic responses following IMT might be influenced by the resistance load, initial respiratory muscle strength, and clinical characteristics of participants[28,35].

Regarding myocardial oxygen consumption, this study did not observe significant differences in RPP between the evaluated conditions. We suggest that IMT does not provoke cardiac overload in subjects with moderate and severe COPD. RPP is often estimated during exercise as a proxy of myocardial requirements as well as tolerance for exercise[36]. In addition, RPP is a strong predictor of cardiovascular and all-cause mortality. These data are in accordance with those in the study of Ramos et al[37] who found no major haemodynamic responses in elderly patients submitted to two sets of IMT sessions at 30% of MIP resisted load. Thus, a single bout of moderate-intensity IMT exercise seems to be safe and well tolerated for moderate to severe COPD patients.

Conclusion

A single session of IMT at 60% of MIP in COPD individuals fails to induce PEH. However, for COPD patients, a short IMT session seem to be clinically safe.

Take home messages

Acute IMT appears to be clinically safe and well tolerated in subjects with COPD and it could be used as an adjuvant exercise to increase patient compliance.

Conflicts of interest

The authors declare no conflict of interest.

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