## Effects of Combined Histamine H1 and H2 Receptor Blockade on Hemodynamic Responses to Dynamic Exercise in Males with High-Normal Blood Pressure

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### Novelty bullets: points that summarize the key findings in the work:
Males with high-normal blood pressure had an exaggerated blood pressure response to exercise. The overactive blood pressure response is known due to an increase in peripheral vasoconstriction. Increase in peripheral vasoconstriction is partially due to inability of histamine receptors.

### Keyword:
histamine receptors, dynamic exercise, peripheral vasoconstriction, mean arterial pressure, cardiac output, total vascular conductance

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Effects of Combined Histamine H\textsubscript{1} and H\textsubscript{2} Receptor Blockade on Hemodynamic Responses to Dynamic Exercise in Males with High-Normal Blood Pressure

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Abstract

While postexercise hypotension is associated with histamine H1 and H2 receptor mediated postexercise vasodilation, effects of histaminergic vasodilation on blood pressure (BP) in response to dynamic exercise are not known. Thus, in 10 normotensive and 10 males with high-normal BP, we examined the effects of histamine H1 and H2 receptor blockade on cardiac output (CO), mean atrial pressure (MAP), aortic stiffness (AoS) and total vascular conductance (TVC) at rest and during progressive cycling exercise. Compared with normotensive group (NMT), MAP, CO, and AoS were higher in the high-normal group (HN) before and after the blockade at rest, while TVC was similar. At the 40% workload, the blockade significantly increased MAP in both groups, while no difference was found in the TVC. CO was higher in the HN than the NMT in both conditions. At the 60% workload, the blockade substantially increased MAP and decreased TVC in the NMT, while no changes in the HN. A similar CO response pattern was observed at the 60% workload. These findings suggest that the mechanism eliciting an exaggerated BP response to exercise in the HN may be partially due to the inability of histamine receptors.

Novelty

● Males with high-normal blood pressure had an exaggerated blood pressure response to exercise

● The overactive blood pressure response is known due to an increase in peripheral vasoconstriction

● Increase in peripheral vasoconstriction is partially due to inability of histamine receptors

Key words: histamine receptors, dynamic exercise, peripheral vasoconstriction, mean arterial pressure, cardiac output, total vascular conductance
Introduction

Recently, the 2018 Hypertension Canada Guidelines released updated guidelines for blood pressure (BP) classifications (Nerenberg et al. 2018). When using non-automated blood pressure, the cut points for “high” are a resting systolic blood pressure (SBP) of > 140 mm Hg and resting diastolic blood pressure (DBP) of > 90 mm Hg and an SBP of 130-139 mm Hg and/or a DBP of 85-89 mm Hg is classified as “high-normal” (Nerenberg et al. 2018). Adults with a BP of 120/80 mm Hg or higher have an increased risk of fatal and nonfatal stroke, ischemic heart disease, noncardiac vascular disease, and doubles the risk for a fatal CVD event (Whelton et al. 2018). Nonpharmacological interventions, such as exercise, are being used in conjunction with pharmacotherapy to lower BP in adults with high-normal blood pressure. Increased physical activity and aerobic exercise participation has been shown to lower SBP in these individuals (Cornelissen and Smart 2013; Garcia-Hermoso et al. 2013; Whelton et al. 2002). Specifically, average reductions in SBP with aerobic exercise is 2 to 4 mm Hg in normotensive adults and 5 to 8 mmHg in hypertensive adults (Cornelissen and Smart 2013), both of which have clinical significance for future CVD risk.

Dynamic exercise evokes an exaggerated BP response in individuals with high-normal compared to normotensive individuals due to increased peripheral vasoconstriction (Choi et al. 2013b; Kim et al. 2014). Higher BP responses during exercise may cause a greater risk of concomitant cardiovascular events such as acute myocardial infarction and sudden death (Mittleman et al. 1993). Understanding the underlying mechanism that causes augmented BP responses during exercise in adults with high-normal is essentially important to reduce future CVD risk.

An exaggerated BP response to exercise may be partly due to altered peripheral vascular responses (Choi et al. 2013b; Kim et al. 2014). It has been suggested that the main physiological response induced by histamine is peripheral vasodilation (Skidgel et al. 2012). Histamine plays an important role in mediating post-exercise hypotension (Romero et al. 2017). Studies have demonstrated that the stimulation of histamine H\textsubscript{1} and H\textsubscript{2} receptors induce peripheral vasodilation which then reduces BP following
exercise (Barrett-O'Keefe et al. 2013; Halliwill et al. 2013; Pellinger et al. 2013). Despite the fact that histamine receptor-mediated peripheral vasodilation contributes to postexercise hypotension, the ability of these receptors to mediate BP responses during exercise is still unknown in individuals with high-normal.

Previously, our study indicated that normotensive sedentary men had an extensive increase in BP responses to exercise due to an increase in peripheral vasoconstriction to submaximal exercise during H₂ receptor blockade (Doh et al. 2016). This finding suggests that histamine receptors may play an important role in mediating vasodilation during exercise and is related to abnormal BP responses in high-normal individuals. Thus, the inability to activate histamine receptors during exercise can augment BP responses due to an increase in peripheral vasoconstriction in these individuals. If this hypothesis is true, the histamine receptors may play a role in inducing an excessive BP response and be a target for a preventive intervention in adults with high-normal BP.

Based on previous findings, we tested the hypotheses that: (1) in normotensive individuals, the excessive BP response during exercise during histamine H₁ and H₂ blockades is primarily due to increased peripheral vasoconstriction; and (2) in individuals with high-normal exaggerated BP responses during exercise is associated with the inability of histamine receptors to reduce peripheral vasoconstriction.
Materials and methods

Twenty recreationally active male participants (10 normotensive SBP < 120 mmHg and DBP < 80 mmHg; 10 high-normal SBP 120-139 and DBP < 80-89 mmHg, both), age 20-27 years, participated in the study. Only male participants were used to avoid potential menstrual cycle effects on exercise hemodynamics. (Choi et al. 2013a). Participants were recruited from the California Baptist University campus. Prior to testing, all participants gave written informed consent form and screened for cardiovascular disease risk factors through a physical activity readiness questionnaire (PAR-Q). All participants were non-smokers and not taking medications that could affect cardiovascular function. They were considered to be in good health and recreationally active. Participants were instructed to abstain from alcohol, caffeine, and strenuous exercise for 24 hours before any intervention. All procedures and protocols used in this study were reviewed and approved by the California Baptist University Institutional Review Board.

Experimental procedures

All participants reported to the laboratory three times over the span of two weeks. On the first visit baseline cardiovascular function was measured. Participants were asked to be seated quietly for 5 min to assess resting heart rate (HR) and BP. Two BP readings were obtained 5 min apart using a mercury sphygmomanometer with the cuff positioned on the left arm at heart level while the participant was seated with their back supported in a chair and both feet on the floor. Two BP measurements were taken and expressed as the average of the two measurements. To determine relative exercise intensity to be used for the subsequent exercise visits, participants completed a VO\(_{2}\text{peak}\) test on a mechanically braked cycle ergometer (Monarch 828, Sweden) using ParvoMedics TrueOne Metabolic System (Sandy, UT). For an accurate VO\(_{2}\text{peak}\), this exercise test was completed in the morning 24-48 hours before submaximal exercise trial. All participants cycled at 0 kp at a pedal cadence of 60 rpm for two minutes and progressively increased work rate by .5 kp every minute until they were exhausted. VO\(_{2}\text{peak}\) was achieved when
participants met all 3 criteria: RER >1.10, VO₂ plateu, and RPE >18. The VO₂peak obtained from this test was used to calculate the relative workload intensities for each participant.

**Exercise protocol**

Participants reported to laboratory for two separate exercise visits. Baseline hydration levels were checked at each visit using specific gravity of urine (ATAGO, Tokyo Japan). After hydration was checked participants rested quietly for 5 min. Resting HR and BP measurements were taken prior to the exercise trial. Following this measurement, participants performed a 12-min bout of submaximal cycling exercise on a mechanically braked cycle ergometer in the absence or presence of combined histamine H₁ and H₂ receptor blockade. Histamine H₁ blockade was induced by fexofenadine 180mg (brand name: Allegra, Pfizer Consumer Healthcare, Morris Plains, NJ, USA) and Histamine H₂ blockade was induced by ranitidine HCL 300mg (brand name: Zantac, Pfizer Consumer Healthcare, Morris Plains, N.J., USA). Participants pedaled at a frequency of 60 r/min during the trial. The exercise protocol started with a warm-up of 2 min at 0 kp, followed by 5 min at 40% VO₂peak and ending with 5 min at 60% VO₂peak. The duration of 40% and 60% of VO₂peak was approximately 5 min for participants to reach at steady state. The same absolute workload was repeated after the blockade in both groups. All hemodynamic data was collected at rest while sitting on the cycle and during exercise. Exercising BP was measured during the last 1 min of 40% and 60% VO₂peak. The washout period was separated by at least 1 week between each test. Fexofenadine takes 2-3 days and ranitidine HCL takes less than 24 hours for complete removal from the body. All exercise tests were performed at the same time of day for each participant. The order of treatment (H₁ + H₂ blockade) was randomly assigned among participants. Participants ingested fexofenadine 1 hour before and ranitidine HCL 2 hours before each protocol because fexofenadine reaches its peak level at about 1.15 h and ranitidine at 2.2 h. Prior to the testing day, participants were given the medication and asked to take them before they came to the laboratory.
Measurement of hemodynamic variables

A noninvasive device was used to measure both HR and stroke volume (SV) via impedance cardiography (Physio Flow, Manatec Biomedical, France) at rest and throughout the experiment. Two electrodes were placed on the left side of the neck, two electrodes were used for recording the ECG, and two electrodes were placed at the xiphoid process. To measure SV, Impedance cardiography detected changes in thorax impedance during cardiac cycle. Cardiac output (CO) was calculated as HR x SVi x BSA, where SVi is the SV index and BSA is body surface area in meters squared. HR was obtained by an ECG using the R-R interval. It has been reported that this impedance technique has been validated and against the Fick method to measure SV and CO (Charloux et al. 2000) and has good reliability at rest and during steady-state moderate and high intensity exercise in healthy people (Gordon et al. 2018). SBP and DBP was measured at the level of the heart from the left arm using a sphygmomanometer at rest and during steady state exercise. Exercising BP was measured by the same investigator in each participant throughout the experiment. The investigator was well trained to measure BP accurately during exercise. In rare case, if the main investigator had difficulty reading it, the BP measurement was confirmed from the other arm using the dual statoscope by two other investigators. MAP was calculated according to the formula MAP = (SBP-DBP)/3 +DBP. Total vascular conductance (TVC) was calculated as CO/MAP. Aortic stiffness (AoS) was calculated by bioelectrical impedance (BI) signals recorded at the chest using impedance cardiography (Physio Flow, Manatec Biomedical, France) at rest (Collette et al. 2011).

Data analysis

The absolute values of the variables measured at rest and during dynamic exercise were compared. Changes in all variables are expressed as means ± SE. Thirty-second averages of HR, SV, CO, TVC, and AoS were taken at rest and last min of exercise at both 40% and 60% of VO2peak and compared between blocked and unblocked conditions. To compare effects of fexofenadine and ranitidine HCL over workloads and between groups a two-way repeated measures ANOVA (SIGMASTAT 4.0, Tulsa OK) and Tukey’s post hoc test was used. Statistical significance was accepted at P < 0.05.
Results

Table 1 indicated physical characteristics of subjects. Young normotensive and high-normal individuals were age matched, but high-normal had a significantly higher body mass index compared to normotensive group. Due to the purpose of this study design, the high-normal group had a significantly higher resting SBP and DBP than the normotensive group ($P = 0.000$ and $P = 0.000$, respectively). Urine specific gravity results before and after the blockade fell between 1.002 and 1.030.

Figure 1 shows the averaged hemodynamic responses at rest before and after histamine H$_1$ and H$_2$ blockade in both groups. Compared with the normotensive group, the high-normal group had a significantly higher HR, MAP, and AoStiff at rest ($P = 0.001$, $P = 0.000$, and $P = 0.011$ respectively). In both conditions, CO was also significantly higher in individuals with high-normal compared with the normotensive individuals ($P = 0.028$ and $P = 0.001$, respectively). There were no significant differences in SV and TVC between both groups compared with before the blockade.

Figure 2 shows the one-minute average changes from rest in SBP, DBP, and MAP during the 40% of VO$_{2peak}$ workload before and after histamine H$_1$ and H$_2$ blockade in both groups. A repeated-measures two-way ANOVA indicated a significant interactive effect (blockade x group) in SBP. SBP had a significant increase in both groups before and after the blockade, but the elevation in SBP was significantly higher after the blockade in the normotensive group (37±3 mmHg vs. 48±4 mmHg) ($P = 0.010$). There was a significant blockade condition effect in MAP. The MAP had significant increase in both groups before and after the blockade, but the elevation in MAP was significantly higher after the blockade ($P = 0.008$). There was no significant difference in DBP before and after the blockade in both groups.

Figure 3 shows one-minute average changes from rest in HR, SV, CO, and TVC during the 40% of VO$_{2peak}$ workload before and after histamine H$_1$ and H$_2$ blockade in both groups. A repeated-measures two-way analysis of variance indicated a significant group effect in SV and CO. 40% exercise intensity
resulted in significant increases in SV and CO in both groups, but the rise in these variables were significantly higher in high-normal group than the normotensive group ($P = 0.050$ and $P = 0.043$, respectively). A repeated-measures two-way ANOVA indicated a significant interactive effect (blockade x group) in HR. The HR was significantly increased with the blockade in only the normotensive group ($P = 0.018$). There was no difference in TVC before and after the blockade in both groups.

Figure 4 shows the one-minute average changes from rest in SBP, DBP, and MAP during the 60% of $\text{VO}_{2\text{peak}}$ workload before and after histamine H1 and H2 blockade in both groups. A repeated-measures two-way ANOVA indicated a significant interactive effect (blockade x group) in both SBP and MAP. SBP and MAP had a significant increase in both groups before and after the blockade, but the elevation in SBP and MAP was significantly higher after the blockade in the normotensive group (SBP: $58\pm3$ mmHg vs. $78\pm3$ mmHg; MAP: $19\pm1$ mmHg vs. $28\pm2$ mmHg) ($P = 0.000$ and $P = 0.000$, respectively). The high-normal group had a significantly higher SBP and MAP response before the blockade compared with the normotensive group ($P = 0.047$ and $P = 0.047$, respectively). There was no significant difference in DBP before and after the blockade in both groups.

Figure 5 shows the one-minute average changes from rest in HR, SV, CO, and TVC during the 60% of $\text{VO}_{2\text{peak}}$ workload before and after histamine H1 and H2 blockade in both groups. A repeated-measures two-way ANOVA indicated a significant group effect in SV and CO. 60% exercise intensity resulted in significant increases in SV and CO in both groups, but the rise in these variables were significantly higher in high-normal group than the normotensive group ($P = 0.050$ and $P = 0.050$, respectively). A repeated-measures two-way ANOVA indicated a significant interactive effect (blockade x group) in TVC. The TVC had a significant decrease in both groups after the blockade, but the decrease in TVC was greater in the normotensive group compared to before the blockade ($150\pm14$ ml/min/mmHg vs. $136\pm12$ ml/min/mmHg) ($P < 0.049$). There was no differences in HR before and after the blockade in both groups.
Discussion

To our knowledge, this is the first study to show the effect of histamine H1 and H2 receptor blockades on BP response during dynamic exercise in individuals with high-normal and normotensive individuals. The major new findings of this study are that the blockades of both histamine H1 and H2 receptors induces the rise in peripheral vasoconstriction and contribute to excessive BP responses in normotensive individuals. As demonstrated previously (Doh et al. 2016), in normotensive individuals the major mechanism to raise BP excessively during exercise in the presence of histamine H2 receptors blockade was primarily due to an increase in peripheral vasoconstriction, with little CO increase. The current study extends these previous observations by demonstrating that the role of both H1 and H2 receptors induces a decrease in peripheral resistance which contributes to BP responses to exercise in normotensive individuals. In contrast, high-normal resulted in significantly higher BP during dynamic exercise compared to normotensive individuals, but the blockades resulted in no significant additional increase in arterial pressure in individuals with high-normal. Taken together, high-normal may decrease the ability of histamine receptors to reduce peripheral vasoconstriction during exercise. Thus, the mechanism that causes excessive BP responses during exercise in individuals with high-normal is partially due to be the inability of histamine receptor H1 and H2 to reduce peripheral vasoconstriction.

Effect of HIGH-NORMALBP on the Hemodynamic Values at Rest

Our results showed that the high-normal had higher arterial blood pressure and heart rate. A previous study reported that, in humans, HR and CO were elevated in high-normal due to sympathovagal imbalance characterized by an elevation in sympathetic activity and a decrease in parasympathetic activity (Davis et al. 2012; Pal et al. 2013). Thus, elevated BP may be induced by a higher CO rather than peripheral vasoconstriction in these individuals. In the present study, we found that CO was significantly elevated in high-normal compared with normotensive individuals at rest, whereas
no difference in TVC was found between two conditions. Thus, the higher BP is likely explained by a higher CO in individuals with high-normal BP.

**Effect of Histamine H<sub>1</sub> and H<sub>2</sub> Receptor Blockade on the Cardiovascular Response during Exercise**

It has been suggested that postexercise hypotension occurs in sedentary and normally active men and women after a bout of aerobic exercise due to peripheral vasodilation mediated by histamine H<sub>1</sub> and H<sub>2</sub> receptors in vasculature (McCord and Halliwill 2006). Recently, a study demonstrated that histamine H<sub>2</sub> receptor blockade augmented the BP response during exercise in normotensive males compared to before the blockade and reported that this phenomenon occurred due to attenuated reduction in peripheral vasoconstriction (Doh et al. 2016). In agreement with this these findings, we investigated that the inability of histamine H<sub>1</sub> and H<sub>2</sub> receptors are responsible for excessive BP responses to exercise in individuals with high-normal BP. Our data have shown that blockade of combined H<sub>1</sub> and H<sub>2</sub> receptors substantially increased arterial BP in normotensive individuals mainly via an increase in peripheral vasoconstriction without any further increase in CO. In contrast, despite the fact that high-normal had an exaggerated BP response before the blockade compared to the normotensives, no difference in BP was found in the absence and presence of the blockade in high-normal individuals. These observations indicate that histaminergic mechanisms play an important role in regulating exercising BP. Thus, in high-normal, excessive BP response to exercise is mediated by marked peripheral vasoconstriction, since the ability of histamine H<sub>1</sub> and H<sub>2</sub> receptors to vasodilate is likely impaired.

One possibility responsible for the peripheral vasoconstriction may be due to the sensitivity of H<sub>1</sub> and H<sub>2</sub> receptors to histamine. It has been demonstrated that a low pH induced by exercise can modify the sensitivity of the H<sub>1</sub> receptor’s binding site to histamine (Ganellin 1982). In this regard, a previous study demonstrated that high-normal had an exaggerated BP to exercise due to an increase in vascular resistance and suggested that this overactive BP response is associated with an augmented reduction in
pH in skeletal muscle (16). Our study adds to the literature by suggesting that changes in vascular resistance related to changes in pH may be related to changes in sensitivity of histamine receptors.

Another explanation for the increased BP responses to exercise is vascular stiffness of large arteries such as the aorta, which was found to be high in high-normal (Gedikli et al. 2010; Karacalioglu et al. 2006). This current study found that individuals with high-normal had higher aortic stiffness compared with the normotensives (figure 1). Aortic stiffness can contribute to increases in peripheral resistance in these individuals (Zhu et al. 2007). Accordingly, the vasculature may be a possible target for interventions that can modify the overactive blood response to exercise and reduce the mortality related to cardiovascular diseases in the future (Lewington et al. 2002).

**Limitations of the Study**

There is evidence that exercise-induced vibration and heat can release histamine from mast cells (Atkinson et al. 1992) and sympathetic withdrawal can cause histamine release (Powell and Brody 1976; Rengo et al. 1978). A limitation to our study is the fact that we did not measure levels of blood histamine during exercise. Previous studies measured whole blood histamine concentrations at rest and at the end of exercise and reported that there were no changes in response to 60 min of submaximal exercise in sedentary, recreationally active men and women (Lockwood et al. 2005; McCord et al. 2006). Likewise, another study showed that there was no significant difference in histamine concentrations during exercise before and after histamine H₂ receptor blockade (Doh et al. 2016). Oh the other hand, a study has been shown to increase histamine release concentrations during short duration of exercise (i.e., 6-12 min) (Campos et al. 1999). The reason for this controversial effect is not clear.

Another issue may be the sympathetic activity, which has been shown to be high in individuals with high-normal (Davis et al. 2012; Pal et al. 2011) and induce histamine release (Powell and Brody 1976; Rengo et al. 1978). Thus, it is assumed that the release of histamine would also affect these...
receptors. We did not measure the recordings of the sympathetic nerve activity in both conditions. Further confirmation needs to be revealed that there is a change in histamine concentrations in the absence and presence of the blockades during a short duration of exercise. Our participants were young, healthy normotensive, and high-normal men and the current findings may not be generalized to other populations.

Clinical Implications

Our findings may have clinical significance and can be useful to high-normal individuals during exercise. This data suggest that high-normal individuals are at a greater risk for concomitant cardiovascular events that can occur in response to exercise via an abnormal BP response (Mittleman et al. 1993). It is important to monitor augmented BP responses relative to the dysfunction of histamine receptors in high-normal individuals during exercise. Thus, the emphasis should be placed on preventative interventions involving the peripheral vascular system.

Conclusions

We demonstrated that the BP response after histamine H$_1$ and H$_2$ receptor blockade is greater during exercise in normotensive individuals and that the contribution of peripheral vasoconstriction to the BP response is substantially increased, whereas there was no difference in high-normal. Thus, this current study suggests that lessened vasodilation by dysfunction of H$_1$ and H$_2$ receptors contributes to an exaggerated BP response to exercise in high-normal.

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Author Disclosure Statement

This manuscript represents original research that has not been for publication elsewhere. All authors have no competing financial interests in relation to the work described, contributed substantially, and approved the final submission.
References


Table Legend

Physical characteristics of subjects.

Values are mean ± standard error; BMI; body mass index, SBP; systolic blood pressure, DBP; diastolic blood pressure, *P < 0.05 vs. normotensive group.
Figure Legends

Fig. 1. Averaged hemodynamic responses at rest before and after histamine H₁ and H₂ blockade in normotensive group (NMT) and group with high-normal (HN). * Between horizontal brackets reflect a significant effect of high-normal blood pressure; # P < 0.05 vs. NMT.

Fig. 2. One-minute average changes from rest in SBP, DBP, and MAP during the 40% of VO₂peak workload before and after histamine H₁ and H₂ blockade in normotensive group (NMT) and group with high-normal (HN). *P < 0.05 vs. control; # P < 0.05 vs. NMT; * Between vertical brackets reflect a significant effect of the blockade.

Fig. 3. One-minute average changes from rest in HR, SV, CO, and TVC during the 40% of VO₂peak workload before and after histamine H₁ and H₂ blockade in normotensive group (NMT) and group with high-normal (HN). *P < 0.05 vs. control; * Between horizontal brackets reflect a significant effect of high-normal blood pressure.

Fig. 4. One-minute average changes from rest in SBP, DBP, and MAP during the 60% of VO₂peak workload before and after histamine H₁ and H₂ blockade in normotensive group (NMT) and high-normal (HN). *P < 0.05 vs. control; # P < 0.05 vs. NMT.

Fig. 5. One-minute average changes from rest in HR, SV, CO, and TVC during the 60% of VO₂peak workload before and after histamine H₁ and H₂ blockade in normotensive group (NMT) and group with high-normal (HN). *P < 0.05 vs. control; * Between horizontal brackets reflect a significant effect of high-normal blood pressure.
Fig 1

- Heart Rate (bpm)
- Stroke Volume (ml)
- Cardiac Output (L/min)
- Mean Arterial Pressure (mmHg)
- Total Venous Compliance (ml/min/mmHg)
- Aortic Stiffness (AU)

Control vs. Blockade comparison for NMT and NH conditions.
Fig 2
Fig 3

- ΔHR (bpm)
- ΔSV (ml)
- ΔCO (L/min)
- ΔTVC (ml/min/mmHg)

Control vs. Blockade comparison for NMT and HN.
Fig 5

- **Δ HR (bpm)**
  - NMT: [Bar chart]
  - HN: [Bar chart]

- **Δ SV (ml)**
  - NMT: [Bar chart]
  - HN: [Bar chart]

- **Δ CO (L/min)**
  - NMT: [Bar chart]
  - HN: [Bar chart]

- **Δ TVC (ml/min/mmHg)**
  - NMT: [Bar chart]
  - HN: [Bar chart]
Table 1. Physical characteristics of subjects.

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<td>23 ± 0.7</td>
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<td>Height (cm)</td>
<td>179 ± 2.7</td>
<td>177 ± 1.9</td>
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<td>Body Weight (kg)</td>
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Values are mean ± standard error; BMI; body mass index, SBP; systolic blood pressure, DBP; diastolic blood pressure. *P < 0.05 vs. normotensive group.