Cardiovascular response to hypoxia after endurance training at altitude and sea level and after detraining

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ENDURANCE EXERCISE TRAINING at altitude or in hypoxia has been reported to induce several physiological adaptations (22, 28, 41), and a number of studies have demonstrated the cardiovascular or ventilatory responses to exercise during the hypoxic condition after altitude training or acclimatization to hypoxia (2, 3, 24, 40). There are a limited number of studies that reported the effects of endurance exercise training during hypoxic conditions on ventilatory response to progressive isocapnic hypoxia (4, 21); these studies indicate that intermittent hypoxic exposure combined with endurance training for several weeks induced an increase in hypoxic ventilatory response (HVR). Hypoxic exposure with or without endurance exercise training may lead to change not only in HVR but also in the cardiovascular response to progressive isocapnic hypoxia. To explore these issue, Insalaco et al. (15) investigated the changes in ventilatory, blood pressure (BP), and heart rate (HR) responses to hypoxia during chronic exposure to an altitude of 5,050 m for 24 days without endurance exercise training. Their study reported an increase in HVR accompanied by an increase in the BP response to progressive isocapnic hypoxia but no change in the HR response, and they suggest that this BP response change is influenced more by ventilation than by chronic exposure to hypoxia (15).

It has hitherto been reported that resting HVR in endurance athletes is lower than that in untrained subjects (6); however, no data are available concerning the effect of endurance training on HVR in normal subjects. Thus it is possible to hypothesize that HVR may decrease after endurance exercise training at sea level. Also, if the BP response to progressive isocapnic hypoxia changes, accompanied by HVR, as proposed by Insalaco et al. (15), it is likely that the BP response to progressive isocapnic hypoxia shows a decrease by endurance training during normoxia and an increase by training during hypoxia. To our knowledge, no study has been made as an attempt to investigate the effect of endurance exercise training in hypoxia or normoxia on cardiovascular response to progressive isocapnic hypoxia.

On the other hand, a number of studies also demonstrated that detraining leads to reductions in maximal and submaximal exercise capacity (7, 29, 31), but the influence of detraining on the cardiovascular response to hypoxia has not been investigated. If the BP response to progressive isocapnic hypoxia is affected by the change in ventilation (15), it is possible to hypothesize that parallel changes in HVR and BP response to progressive isocapnic hypoxia occur not only during endurance training in hypoxia or normoxia but also during detraining.
The purpose of this study was, therefore, to elucidate 1) the influence of endurance training at 4,500 m and at sea level and of detraining on ventilatory and cardiovascular responses to progressive isocapnic hypoxia and 2) whether the change in cardiovascular response is correlated to the change in ventilatory response after endurance training in either hypoxia or normoxia and during detraining. The results in the present study should provide further elucidation of the ventilatory and cardiovascular adaptations or regulation to subsequent hypoxic exposure after endurance training at altitude or sea level and during detraining. Furthermore, these data could give insight into the relationship between the ventilatory and cardiovascular adaptations to progressive isocapnic hypoxia in humans.

METHODS

This study was completed in conjunction with a study designed to clarify the effects of endurance training and detraining on ventilatory chemosensitive adaptations (18). It was approved by the Human Research Committee of the Research Center of Health, Physical Fitness and Sports of Nagoya University.

Experimental procedures. The procedures used in this study were described fully in our previous study (18) and will be outlined here briefly. Fourteen healthy male volunteers were assigned to an altitude training group (ATG) or a sea-level training group (SSTG). The hypobaric chamber was maintained at 432 Torr, corresponding to an altitude of 4,500 m. The altitude training group were assigned to an altitude of 4,500 m and were trained for 30 min/day, 5 days/wk, for 2 wk. V\(_{\text{O}2}\)max and ventilatory exercise training at sea level with intensity corresponding to 70% of V\(_{\text{O}2}\)max measured at sea level. The hypoxic chamber was used for endurance exercise training in the altitude training group. The pressure of the hypoxic chamber was maintained at 432 Torr, corresponding to an altitude of 4,500 m. The altitude training group underwent endurance training at the same relative exercise intensity as the sea level group (70% of altitude V\(_{\text{O}2}\)max). The subjects in both groups trained on a mechanical bicycle ergometer (Monark) with a frequency of 60 rpm. Both groups trained for 30 min/day, 5 days/week, for 2 wk. V\(_{\text{O}2}\)max and ventilatory and cardiovascular responses to progressive isocapnic hypoxia were measured at sea level for both groups before (Pre) and after endurance training. The posttraining test was performed twice, i.e., immediately after exercise training for 2 wk (Post) and after 2 wk of detraining (Det).

Ventilatory and cardiovascular responses to hypoxia. The HVR at sea level was measured by using a progressive isocapnic hypoxic test proposed by Weil et al. (42). A rebreathing system was used similar to that of the previous study (17). During the HVR test, tidal volume (VT), inspiratory duration (TI), expiratory duration, end-tidal CO\(_2\) and O\(_2\) fraction (PET\(_{\text{CO}_2}\) and PET\(_{\text{O}_2}\)), arterial oxygen saturation (SaO\(_2\)), arterial BP, and ECG signals were digitized at a sampling rate of 100 Hz through analog-to-digital conversion (ADX-989, Canopus). The digitized signals were stored in a computer (PC-9821XA, NEC). To eliminate cardiovascular variability with respiratory cycle and to compare cardiovascular response with ventilation, HR and BP values were measured beat to beat and averaged in a breath-by-breath fashion (15). The FVTI, SBP, DBP, and ECG signals were drawn through a sampling tube connected to the mouthpiece and was analyzed by a gas analyzer (type MG-360, Minato Ikagaku) to measure PET\(_{\text{CO}_2}\) and PET\(_{\text{O}_2}\). The end-tidal partial pressure of CO\(_2\) and O\(_2\) (PET\(_{\text{CO}_2}\) and PET\(_{\text{O}_2}\)) were calculated from PET\(_{\text{CO}_2}\) and PET\(_{\text{O}_2}\), respectively. SaO\(_2\) was measured by using a finger pulse oximeter (OLV-1200, Nihon Koden). Systolic BP (SBP) and diastolic BP (DBP) were continuously recorded with a Finapres BP monitor (model 2300, Ohmeda). The probe of the pulse oximeter and the finger cuff of the Finapres were kept constant at heart level. HR was calculated from every R-R interval obtained from the ECG. The respiratory flow, PET\(_{\text{CO}_2}\), PET\(_{\text{O}_2}\), SaO\(_2\), BP, and ECG signals were stored in a computer (PC-9821XA, NEC). To eliminate cardiovascular variability with respiratory cycle and to compare cardiovascular response with ventilation, HR and BP values were measured beat to beat and averaged in a breath-by-breath fashion (15).

Statistical analysis. Values are expressed as means ± SD. The differential changes in parameters during the experimental periods between the altitude and sea-level training groups were compared by using the two-way ANOVA with repeated measurements. Differences in the parameters at each session (Pre, Post, and Det) within each group were determined by using the Wilcoxon test, and the comparison of parameters between groups at each session was done using the Mann-Whitney test. The relationships among the parameters were determined by a simple linear regression analysis. The SPSS statistical package (SPSS, Chicago, IL) was used for these analyses. Statistical significance was defined as P < 0.05.

RESULTS

HVR. Resting ventilation, f, VTTI, PET\(_{\text{O}_2}\), and PET\(_{\text{CO}_2}\) did not change in both groups throughout the experimental period (18).

There were no significant differences in the HVR between the altitude training group and the sea-level training group before training. In the altitude training group, the HVR showed an insignificant increase after combined intermittent hypoxic exposure with endurance training for 2 wk [0.49 ± 0.22 (Pre) to 0.67 ± 0.22 (Post)] l·min\(^{-1}\)·%\(^{-1}\). On the other hand, after endurance exercise training at sea level, a significant (P < 0.05) decrease in the HVR was found in the sea-level training group [0.43 ± 0.22 (Pre) to 0.25 ± 0.19 (Post)] l·min\(^{-1}\)·%\(^{-1}\). During 2 wk of detraining, the changed HVR after endurance training in both groups were restored [0.42 ± 0.23 l·min\(^{-1}\)·%\(^{-1}\) (Det) in the altitude training group, 0.37 ± 0.23 l·min\(^{-1}\)·%\(^{-1}\) (Det) in the sea-level training group], as mentioned in the previous study (18).

Similar to HVR, in the altitude training group the Δf/ΔSaO\(_2\), and ΔVTI/ΔSaO\(_2\) tended to increase, but insignificantly, after endurance training during hypoxia, and the changed Δf/ΔSaO\(_2\), and ΔVTI/ΔSaO\(_2\) were restored to pretraining level during 2 wk of detraining [Δf/ΔSaO\(_2\): 0.14 ± 0.09 (Pre), 0.19 ± 0.13 (Post), 0.11 ± 0.12 (Det) breaths·min\(^{-1}\)·%\(^{-1}\); ΔVTI/ΔSaO\(_2\): 16.6 ± 6.4 (Pre), 21.1 ± 11.6 (Post), 11.9 (Det) ml·s\(^{-1}\)·%\(^{-1}\). In
contrast, the $\Delta f/\Delta SaO_2$ and $\Delta (V_{T}/T_{I})/\Delta SaO_2$ showed a significant decrease ($P < 0.05$) in the sea-level training group after training for 2 wk, and these parameters were restored during detraining ($\Delta f/\Delta SaO_2; 0.14 \pm 0.11$ (Pre), 0.08 $\pm$ 0.09 (Post), 0.13 $\pm$ 0.11 (Det) breaths·min$^{-1}$·%$^{-1}$; $\Delta (V_{T}/T_{I})/\Delta SaO_2; 14.1 \pm 6.8$ (Pre), 7.6 $\pm$ 7.5 (Post), 12.0 $\pm$ 8.6 (Det) mm$^3$·s$^{-1}$·%$^{-1}$).

Cardiovascular responses to progressive isocapnic hypoxia. Resting SBP, DBP, and HR did not change significantly in both groups throughout the experimental period, as shown in Table 1.

Figure 1 indicates resting SBP, DBP, and HR responses to progressive isocapnic hypoxia at Pre, Post, and Det in both groups. $\Delta$SBP/$\Delta SaO_2$, $\Delta$DBP/$\Delta SaO_2$, and $\Delta$HR/$\Delta SaO_2$ were not significantly different between the groups before endurance training. Mean values of $\Delta$SBP/$\Delta SaO_2$ increased significantly ($P < 0.05$) after endurance training with hypoxic exposure in the altitude training group [0.67 $\pm$ 0.32 (Pre) to 1.05 $\pm$ 0.35 (Post) mmHg/%]. By contrast, in the sea-level training group the $\Delta$SBP/$\Delta SaO_2$ decreased significantly ($P < 0.05$) from 0.51 $\pm$ 0.45 (Pre) to 0.21 $\pm$ 0.49 (Post) mmHg/%. There was a significant difference ($P < 0.05$) in $\Delta$SBP/$\Delta SaO_2$ measured after training (Post) between the altitude training group and the sea-level training group (Fig. 1A). After detraining for 2 wk, the changed $\Delta$SBP/$\Delta SaO_2$ in both groups returned to pretreatment values as shown in Fig. 1A [altitude training group, 0.59 $\pm$ 0.51 mmHg/% (Det); sea-level training group, 0.62 $\pm$ 0.57 mmHg/% (Det)]. There was a significant difference in $\Delta$SBP/$\Delta SaO_2$ between the groups during the experimental period ($\bar{F} = 8.75, P < 0.05$).

Mean $\Delta$DBP/$\Delta SaO_2$ values determined at Pre, Post, and Det were 0.10 $\pm$ 0.33, 0.20 $\pm$ 0.34, and 0.14 $\pm$ 0.52 mmHg/% for the altitude training group and 0.10 $\pm$ 0.30, $0.01 \pm 0.13$, and 0.08 $\pm$ 0.27 mmHg/% for the sea-level training group, respectively. As shown in Fig. 1B, there were no significant changes in the $\Delta$DBP/$\Delta SaO_2$ in either the altitude training group or the sea-level training group throughout the experimental period.

The $\Delta$HR/$\Delta SaO_2$ did not show any changes after endurance training and detraining over 2 wk in both groups (Fig. 1C), i.e., 0.85 $\pm$ 0.36 (Pre), 0.79 $\pm$ 0.36 (Post), and 0.87 $\pm$ 0.28 (Det) beats·min$^{-1}$·%$^{-1}$ in the altitude training group and 0.83 $\pm$ 0.43 (Pre), 0.82 $\pm$ 0.30 (Post), and 0.90 $\pm$ 0.45 (Det) beats·min$^{-1}$·%$^{-1}$ in the sea-level training group.

Comparison of ventilatory and cardiovascular responses. The magnitude of the changes in the HVR ($\delta$HVR, l·min$^{-1}$·%$^{-1}$), $\Delta$SBP/$\Delta SaO_2$ ($\delta$SBP/$\Delta SaO_2$, mmHg%), $\Delta$DBP/$\Delta SaO_2$ ($\delta$DBP/$\Delta SaO_2$, mmHg%), and $\Delta$HR/$\Delta SaO_2$.
(ΔΔHR/ΔSaO₂, beats·min⁻¹·%⁻¹) was calculated as the difference (Δ) between those obtained before and after endurance training (Pre – Post) and after endurance training and after detraining (Post – Det). In comparison to the changes in ventilatory and cardiovascular responses to hypoxia after endurance training and detraining, there were significant correlations between the ΔHVR and ΔΔSBP/ΔSaO₂ by endurance training (Pre – Post, r = 0.51, P < 0.05) and by detraining (Post – Det, r = 0.63, P < 0.05), as shown in Fig. 2A; however, no correlation of ΔHVR with either ΔΔDBP/ΔSaO₂ or ΔΔHR/ΔSaO₂ was found (Fig. 2B and C).

DISCUSSION

The objectives of this study were twofold: 1) to elucidate the changes in ventilatory and cardiovascular responses to progressive isocapnic hypoxia after endurance training during hypoxia and normoxia and during detraining and 2) to clarify whether the change in the cardiovascular responses to hypoxia is correlated to the change in HVR. We found that 1) SBP response to progressive isocapnic hypoxia changed significantly in parallel to HVR after endurance training in hypoxic or normoxic condition and during detraining, i.e., ΔSBP/ΔSaO₂ and HVR showed an increase in the altitude training group and a decrease in the sea-level training group after endurance training for 2 wk, and the changed ΔSBP/ΔSaO₂ and HVR were restored to the pretraining level in both groups during 2 wk of detraining; 2) the DBP and HR responses to isocapnic progressive hypoxia did not indicate significant changes after endurance training and during detraining in both groups, and 3) significant correlations were observed between ΔHVR and ΔΔSBP/ΔSaO₂ by endurance training (Pre – Post, r = 0.51, P < 0.05) and by detraining (Post – Det, r = 0.63, P < 0.05), respectively. As far as we know, this is the first study to evaluate the effects of endurance exercise training at altitude and at sea level, as well as those of detraining, on BP and HR responses to isocapnic progressive hypoxia.

Although the ventilatory chemosensitive adaptations during chronic hypoxic exposure have been reported by numerous studies (11, 12, 32, 33, 35, 43), the influence of hypoxic exposure on cardiovascular response to isocapnic progressive hypoxia has received little attention. To our knowledge, no study has examined the influence on ventilatory, BP, and HR responses to hypoxia of a sojourn to an altitude without endurance training; nevertheless, ΔΔHR/ΔSaO₂ increased in parallel to HVR as shown in Fig. 1.

Several possibilities may explain the discrepancy in the observed DBP response between the present study

Fig. 2. Relationship between changes in hypoxic ventilatory response (ΔHVR) with changes in SBP (ΔΔSBP/ΔSaO₂, A), DBP (ΔΔDBP/ΔSaO₂, B), and HR responses (ΔΔHR/ΔSaO₂, C) to progressive isocapnic hypoxia during endurance training (at Pre and Post) and detraining (at Post and Det) periods.
and that of Insalaco et al. (15). First, our experimental procedures for the altitude training group differed partially from those of Insalaco et al., e.g., endurance exercise training (with vs. without), procedure of altitude exposure (intermittent vs. chronic), exposure period (2 wk vs. 24 days), and location of the measurements (sea level vs. 5,050 m). Second, a difference in cardiac adaptation with or without endurance training may exist in the mechanisms. Liu et al. (23) studied the effect of endurance training at altitude on the resting cardiac functions at sea level in athletes and indicated an elevated cardiac systolic function and cardiac output at rest after altitude training. On the other hand, resting cardiac output in acclimated subjects at sea level is lower than that in unacclimated subjects (19). Therefore, different changes in the cardiac adaptation after training during hypoxia may have affected our results. Third, differences in the sympathetic responses to hypoxia after hypoxic exposure with or without endurance training could also be a contributing factor. Prior investigations have shown that chronic exposure to continuous hypoxia leads to increased sympathetic activity (1, 27, 44), and this increased sympathetic activity had a relation to an elevation in systemic arterial BP (27, 44). Also, Greenberg et al. (13) examined the effect of chronic intermittent hypoxia on sympathetic activity and arterial BP response to subsequent chemoreflex stimulation in animals, and they indicated that chronic intermittent hypoxia for 30 days increased both sympathetic responsiveness and systemic arterial pressure response to chemoreflex stimulation. Judging from these data, sympathetic activity to subsequent hypoxic stimulation in the altitude training group in the present study may have been modulated after the combined endurance training and intermittent hypoxic exposure. However, in the present study, increased arterial BP at rest was not observed after endurance training during hypoxia, and the degree of increases in HVR and ΔSBP/ΔSaO₂ observed after endurance exercise training during hypoxia was smaller than that reported by Insalaco et al. (15). Therefore, we speculate that the exposure period of endurance training during intermittent hypoxia 30 min/day, 5 days/wk, for 2 wk, as applied here, may have been insufficient duration to alter DBP responses to subsequent hypoxia, and differences in the degree and duration of hypoxic exposure might also explain differences in the magnitude of the changes in ΔDBP/ΔSaO₂ between the study of Insalaco et al. (15) and the altitude training group of the present one. In other words, it is possible to speculate that ΔDBP/ΔSaO₂ may increase significantly after intermittent hypoxic exposure with endurance training, as the periods are prolonged.

In contrast to the results of increases in HVR and ΔSBP/ΔSaO₂ in the altitude training group, HVR and ΔSBP/ΔSaO₂ in the sea-level training group did decrease significantly, whereas there was no significant change in the ΔDBP/ΔSaO₂ after endurance training at sea level over 2 wk. Similar to the altitude training group, a few factors may be responsible for the unchanged ΔDBP/ΔSaO₂ despite significantly decreased ΔSBP/ΔSaO₂, e.g., resting cardiac adaptations and activity sympathetic to progressive isocapnic hypoxia after endurance training at sea level. In conjunction with endurance training in hypoxia and normoxia, we determined ventilatory and cardiovascular responses to progressive isocapnic hypoxia during detraining. Interestingly, after 2 wk of detraining, the changed HVR and ΔSBP/ΔSaO₂ did return to their levels from before the endurance training in both groups (Fig. 1A), whereas there was no significant change in ΔDBP/ΔSaO₂ in either the altitude or the sea-level training group during 2 wk of detraining (Fig. 1B). These results suggest that the SBP response to progressive isocapnic hypoxia is more variable than that of the DBP response not only during endurance training either at altitude or sea level but also during detraining, similar to hypoxic ventilatory chemosensitivity for short periods. Because we could not determine other parameters such as sympathetic activity and cardiac output in this study, the mechanism for differences of changes in ΔSBP/ΔSaO₂ and ΔDBP/ΔSaO₂ cannot be adequately discussed. Further research is required to elucidate the mechanisms of this phenomenon.

It is interesting to note that there were significant correlations between ΔHVR and ΔSBP/ΔSaO₂ by endurance training (Pre–Post, r = 0.51, P < 0.05) and by detraining (Post – Det, r = 0.63, P < 0.05) as shown in Fig. 2A but not ΔDBP/ΔSaO₂ (Fig. 2B). Insalaco et al. (15) also demonstrated significant correlations between absolute values of HVR and ΔBP/ΔSaO₂ throughout a sojourn at high altitude and concluded that these significant relationships give evidence of a strong influence of ventilation on the BP. It is conceivable that changed HVR in both groups after endurance training and detraining reflects the changing drive from the carotid body chemoreceptor. Ventilation and sympathetic activity are simultaneously increased by acute hypoxia; thus links exist between the ventilatory and sympathetic responses to acute hypoxia (10, 39). On the other hand, several reports have indicated that the increase in systemic arterial pressure during chronic hypoxic exposure is related to the degree of sympathetic activity that seems to be activated more by concomitant hyperventilation than by hypoxia per se (1, 44). From the studies above and the results in this study, it is likely that the changes in ventilatory response to hypoxia by training and detraining relate to the changes in SBP response in the present study. Besides the ventilation, several mechanisms’ interactions influence BP responses (8, 14, 39). It is generally accepted that systemic arterial BP is maintained primarily by the carotid chemoreceptor reflex to hypoxia, i.e., the pressor responses, which are caused by vasoconstriction in skeletal muscle and several other vascular beds and by increasing cardiac output (9, 26, 39). By contrast, these pressor responses are opposed by depressor effects arising from activation of pulmonary afferents by hyperventilation and by the local vasodilation due to the direct action of hypoxia on peripheral vascular beds (9, 14, 26, 39). Also, there is an interaction between the baroreceptors and the chemoreflex...
responses to hypoxia (39). Concerning hemodynamic response to progressive isocapnic hypoxia developed in parallel to ventilatory response, Serebrovskaya (37) assumed that parallel reflex reactions of respiration and circulation may be induced by the impulses from the peripheral chemoreceptors sensitive to the hypoxia simultaneously reaching the respiratory and vasomotor centers. From several reports mentioned above, because the mechanism of BP response to hypoxia is complicated and the significant correlations between ∆HVR and ∆SBP/∆SaO2 by endurance training and detraining cannot establish cause and effect, it cannot be proved that the changes in the SBP response in the present study are simply induced by the changes in ventilatory response to hypoxia as proposed by Insalaco et al. (15), but it seems reasonable to suppose that there is an interaction between the changes in the SBP and ventilatory responses to isocapnic hypoxia by endurance training and detraining.

It is well known that resting HR in endurance athletes is lower than that in high-altitude climbers or in untrained subjects (6, 34, 36), whereas the effects on HR response to progressive isocapnic hypoxia have been scarcely studied. The cross-sectional study by Slutsky and Rebuck (38) demonstrated that HR response to progressive isocapnic hypoxia does not correlate to HVR in humans. Ohyabu et al. (30) determined the ventilatory and HR responses to progressive isocapnic hypoxia in athletes and nonathletes. In their study, the HVR in long-distance runners was significantly lower than that of the sedentary subjects, whereas HR response to hypoxia was almost the same in both groups. These studies may support the results of the present study, in which the HR response did not change after endurance training in the sea-level training group, despite the significant decrease in HVR (Fig. 1C).

Moreover, the longitudinal study (15) has reported significant increases in HVR and BP responses to progressive isocapnic hypoxia, but not in the HR response, after chronic exposure to high altitude. Our study found that the altitude training group did not show a change in ∆HR/∆SaO2 after training during hypoxia (Fig. 1C). The present data are in agreement with the previous study (15). These results indicate that there is no change in HR response to progressive isocapnic hypoxia, even if HVR and BP responses to hypoxia do increase or decrease by endurance training with or without hypoxic exposure.

At present, several factors may be responsible for the absence of alteration in HR responses to hypoxia (14, 20, 38), although it is difficult to explain this on the basis of the physiological grounds of the HR response to isocapnic progressive hypoxia obtained here. The carotid chemoreceptor reflex to hypoxia leads to a slowing of HR, whereas the hyperventilation induced by hypoxia is a result of cardioaccelerator reflexes through lung inflation receptors (9, 14, 26). The aortic chemoreceptors, in contrast to the carotid chemoreceptors, may cause tachycardia rather than bradycardia (16). The increase in arterial BP resulting from hypoxic exposure may also stimulate the baroreflexes and could contribute to modifying the HR response (20, 25). In addition, autonomic adaptation may be included in the unchanged HR response to hypoxia. Previous study reported that HR in acclimatized subjects during acute exposure to altitude is lower than that in nonacclimatized subjects (19), and other studies concluded that this blunting response to hypoxia is related to β-receptors’ downregulation with adaptation to high altitude (27, 44). Thus β-receptors’ downregulation may relate to the unchanged HR response to isocapnic hypoxia after endurance training during hypoxia. Conversely, it has been reported that endurance training at sea level induces change in autonomic activity of the cardiovascular regulation system (5). Accordingly, we cannot exclude an effect of autonomic activity, including sympathetic and parasympathetic activity, and β-adrenergic mechanisms after training on the HR response to hypoxia, although we did not show evidence indicating alteration of autonomic activity after endurance training at sea level. Taking these observations into consideration, it is possible to assume that the lack of changes in HR responses in both groups may be results of the fact that those opposite effects, i.e., accelerating and braking effects on HR, offset each other (15). However, it is necessary to investigate further to confirm this assumption.

In conclusion, SBP response to progressive isocapnic hypoxia increased significantly after endurance training during hypoxia and decreased significantly after endurance training at sea level for 2 wk, and these changed SBP responses in both groups were restored to pretraining levels in a parallel fashion with HVR during 2 wk of detraining. Neither DBP nor HR responses changed significantly after endurance training and during detraining. There were significant correlations between the changes in the HVR and ∆SBP/∆SaO2 by endurance training and detraining. These results suggest that the SBP response to isocapnic hypoxia is variable after endurance training in hypoxic or normoxic conditions and during detraining for short periods, as is the ventilatory response to hypoxia, but not DBP and HR responses. They also suggest that there is an interaction between the changes in the SBP and the ventilatory responses to progressive isocapnic hypoxia after endurance training or during detraining.

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