Intermittent hypobaric hypoxia exposure does not cause sustained alterations in autonomic control of blood pressure in young athletes

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ENDURANCE ATHLETES OFTEN USE acclimatization at altitude to improve sea-level performance (13). This strategy, in which the altitude exposure is combined with intermittent normoxia for training purposes, the so-called “living high-training low” model, has been demonstrated to be particularly effective and is becoming widespread among endurance athletes (9, 10, 31, 38, 46, 47). In an attempt to make this type of altitude training more efficient and more accessible to a larger number of athletes who do not live in mountainous regions, many investigators have used models of artificial environments, such as hypoxic breathing devices or hypobaric chambers, to examine progressively shorter exposures to simulated high altitude. In these models, the hypoxic exposure may be as short as a few minutes to a few weeks (33, 40, 54). Variations of intermittent hypoxia (IH) are also used in animals as a model of sleep apnea, causing sustained sympathoexcitation and hypertension in animals and, thus, raising concerns about the safety of this model. However, cardiovascular and autonomic manifestations of IH are less well characterized than continuous hypoxemia and are controversial (8, 22). It remains unclear whether IH alters the autonomic nervous system in competitive athletes.

The present study was therefore performed to test the hypothesis that exposure to IH for 4 wk affects autonomic control of blood pressure (BP) in healthy trained young individuals. To accomplish this objective, steady-state hemodynamics were measured, dynamic cardiovascular regulation was assessed by spectral and transfer function analysis of cardiovascular variability, and cardiac-vagal baroreflex function was evaluated by a Valsalva maneuver twice before and 3 days after the last chamber exposure. We found no significant differences in HR, BP, Qc, SV, TPR, cardiovascular variability, or cardiac-vagal baroreflex function between the groups at any time. These results suggest that exposure to intermittent hypobaric hypoxia for 4 wk does not cause sustained alterations in autonomic control of BP in young athletes. In contrast to animal studies, we found no secondary evidence for sustained physiologically significant sympathoexcitation in this model.

altitude; autonomic nervous system; hemodynamics; arterial pressure; cardiovascular variability; baroreflexes

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had not been at
throughout the intervention. The athletes were sea-level residents and
typical volume and intensity and continued a similar training schedule
/H11021
2:18 (min:s) for men and women, respectively. In the 3 mo before
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the normoxic group, the first 10 min of exposure involved multiple
pressed and decompressed to provide multiple pressure changes. For
None of the subjects had substantive experience with high altitude
No exercise was undertaken while the subjects were in the chamber.
The study consisted of three phases: the pretesting phase, the 4-wk
chamber exposure, and the posttesting phase. All subjects underwent
the intervention for data analysis. Only the chamber technicians were aware of the
treatment for each group, and all subjects and investigators were blinded until the end of the experiment and data-checking phase.

Experimental protocols. All experiments were performed with
subjects in the sitting position, in the morning, ±2 h after a light
breakfast and >12 h after the last caffeinated or alcoholic beverage,
in a quiet, environmentally controlled laboratory with an ambient
AJP-Regul Integr Comp Physiol • VOL 292 • MAY 2007 • www.ajpregu.org

Table 1. Subject characteristics

<table>
<thead>
<tr>
<th>Variable</th>
<th>IH (n = 10)</th>
<th>Normoxia (n = 12)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, yr</td>
<td>22.5±7.3</td>
<td>22.8±9.3</td>
<td>0.928</td>
</tr>
<tr>
<td>Sex, male/female</td>
<td>46/5</td>
<td>7/5</td>
<td></td>
</tr>
<tr>
<td>Height, cm</td>
<td>171±8</td>
<td>175±10</td>
<td>0.324</td>
</tr>
<tr>
<td>Mass, kg</td>
<td>66±13</td>
<td>67±12</td>
<td>0.843</td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td>22.5±2.7</td>
<td>21.9±2.2</td>
<td>0.553</td>
</tr>
<tr>
<td>Sports type, runner/swimmer</td>
<td>5/5</td>
<td>5/7</td>
<td></td>
</tr>
<tr>
<td>Hematocrit, %</td>
<td>40.9±3.7</td>
<td>41.5±2.9</td>
<td>0.696</td>
</tr>
<tr>
<td>Peak O₂ uptake, ml·min⁻¹·kg⁻¹</td>
<td>55.2±9.6</td>
<td>55.8±7.1</td>
<td>0.859</td>
</tr>
</tbody>
</table>

Values are means ± SD. BMI, body mass index; IH, intermittent hypoxia.
Comparisons between groups were made using unpaired t-tests.
strain and ending at the nadir of SBP during the 15-s straining period. The slope of the relation between the reductions in SBP and the corresponding decreases in RRI was determined for each subject by least-squares linear regression analysis (21) and used to evaluate vagal baroreflex sensitivity.

Cardiac-vagal baroreflex sensitivity was also assessed for each subject during phase IV of the Valsalva maneuver, which is identified as the time from the first increase in SBP after the release of Valsalva maneuver straining to the first noticeable pressure drop after overshoot (45). The slope of the linear correlation between the elevations in SBP and the corresponding increases in RRI was examined to evaluate cardiac-vagal baroreflex sensitivity (21).

Additionally, the baroreflex-mediated sympathetic vasoconstrictor response was evaluated from the difference between the highest SBP during phase IV (i.e., overshoot) and the averaged SBP at baseline. The Valsalva straining-produced reduction in BP was quantified as the response was evaluated from the difference between the highest SBP during phase IV of the Valsalva maneuver, which is identified as the time from the first increase in SBP after the release of Valsalva maneuver straining to the first noticeable pressure drop after overshoot (45). The slope of the linear correlation between the elevations in SBP and the corresponding increases in RRI was examined to evaluate cardiac-vagal baroreflex sensitivity (21).

RESULTS

Two subjects, one in each group (intervention and placebo), complained of a headache and nausea and were treated with acetaminophen (Tylenol), and one subject in each group complained of sinus pressure, which was relieved by physical maneuvers. Oxyhemoglobin saturation data confirmed prominent hypoxemia in the IH group, which was not present in the normoxic group before (Table 1) and after chamber exposure: hematocrit (23) and peak oxygen uptake (39) changed after chamber exposure, and neither parameter was different between the IH and the normoxic group before (Table 1) and after chamber exposure: hematocrit = 41.6 ± 2.9 vs. 40.1 ± 3.3% (P = 0.28) and peak oxygen uptake = 56.1 ± 8.4 vs. 55.1 ± 6.6 ml·min⁻¹·kg⁻¹ (P = 0.76).

Table 2. Oxyhemoglobin saturation during chamber exposure

<table>
<thead>
<tr>
<th>Chamber Exposure</th>
<th>IH (n = 10)</th>
<th>Normoxia (n = 12)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Day 5</td>
<td>68.2 ± 9.3</td>
<td>97.7 ± 1.2</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Day 8</td>
<td>69.8 ± 8.9</td>
<td>97.9 ± 1.2</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Day 15</td>
<td>68.1 ± 4.6</td>
<td>97.5 ± 0.9</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Day 18</td>
<td>64.7 ± 9.0</td>
<td>97.6 ± 0.9</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Values are means ± SD. Comparisons between groups and days of chamber exposure were made by 2-way repeated-measures ANOVA on rank, with Bonferroni’s post hoc pairwise multiple comparison analysis.

Steady-state hemodynamics. Table 3 displays resting steady-state hemodynamics before and after chamber exposure in both groups. In the resting, seated position, SBP (P = 0.62), DBP (P = 0.32), HR (P = 0.91), Q˙c (P = 0.76), SV (P = 0.61), and TPR (P = 0.81) during the two pretests before chamber exposure were not different between the two groups. These hemodynamic variables were not different between the groups after chamber exposure (Table 3; P = 0.80 for SBP, 0.85 for DBP, 0.71 for HR, 0.50 for Q˙c, 0.20 for SV, and 0.38 for TPR). Moreover, for the IH and normoxic groups, SBP (P = 0.34 and 0.21, respectively), DBP (P = 0.16 and 0.21), HR (P = 0.63 and 0.39), Q˙c (P = 0.72 and 0.78), SV (P = 0.79 and 0.27), and TPR (P = 0.93 and 0.32) remained unchanged after chamber exposure within the group. Statistical power was calculated on the basis of the two pretest measurements. In general, power was 0.80–0.90 for most measurements within groups; however, for some variables, power was less: 0.889 for DBP in the IH group and 0.182 for SBP in the normoxic group.

Dynamic cardiovascular regulation. Table 4 summarizes the indexes of cardiovascular variability and baroreflex sensitivity. Before chamber exposure, the low- and high-frequency power of RRI variability (LFRR and HFRR) were not different between the groups (P = 0.52 and 0.33 for LFRR and HFRR, respectively). Also, the normalized power in LFRR (P = 0.91) and HFRR (P = 0.31) was not different between the groups. Similarly, the low- and high-frequency power of SBP variability (LFBP and HFBP) did not differ between the groups (P = 0.67 and 0.21 for LFBP and HFBP). Gain LF and Gain HF were similar in the IH and normoxic groups before chamber exposure (Table 3; P = 0.90 and 0.97). Moreover, none of these indexes, except normalized LFRR in the IH group, was different between the two tests before the chamber exposure within the group (Table 4).

After 4 wk of chamber exposure, the indexes of cardiovascular variability (except LFBP) remained unchanged in all subjects (Table 4), and these variables did not differ between the groups (P = 0.41 for LFRR, 0.68 for HFRR, 0.91 for normalized LFRR, 0.54 for normalized HFRR, 0.33 for LFBP, and 0.55 for HFBP), indicating that chronic intermittent hypoxic hypoxia had no influence on cardiovascular variability in these young athletes. Gain LF and Gain HF did not change in either group (Table 4), suggesting an unaltered baroreflex function after chamber exposure.

Cardiac-vagal baroreflex function. Figure 1A shows original traces of arterial pressure and HR during the Valsalva maneuver from one representative subject. The linear correlation between RRI and the corresponding SBP was determined during early phase II (i.e., BP-decreasing period) and phase IV (i.e., BP-increasing period) of the Valsalva maneuver, and the slope of the line indicates the cardiac-vagal baroreflex sensitivity (Fig. 1B). Before chamber exposure, cardiac-vagal baroreflex sensitivity was similar between the groups during decreasing BP (Fig. 2A; P = 0.48) or during increasing BP (Fig. 2B; P = 0.28). Cardiac-vagal baroreflex sensitivity remained unchanged after chamber exposure in both groups (Fig. 2A; P = 0.41 and 0.98 for IH and normoxic groups, respectively; Fig. 2B, P = 0.36 and 0.70), suggesting that chronic IH did not affect cardiac-vagal baroreflex function in these healthy young athletes during decreases or increases in arterial pressure.
Sympathetic vasocostrictor response. Valsalva straining produced a reduction in BP, quantified as the difference between the highest DBP during phase I and the lowest DBP during early phase II, which was not different between the groups before chamber exposure: 21 ± 9 and 23 ± 12 mmHg in the IH and normoxic groups, respectively, during pretesting 1 (P = 0.71) and 19 ± 8 and 16 ± 8 mmHg in the IH and normoxic groups, respectively, during pretesting 2 (P = 0.48). It was also not different between the groups after chamber exposure: 23 ± 10 and 27 ± 11 mmHg in the IH and normoxic groups, respectively (P = 0.46).

The difference between the highest SBP during phase IV (i.e., BP overshoot) and the averaged SBP at baseline, which was used as an index of sympathetic vasocostriction, was not different between the groups before chamber exposure: 26 ± 15 and 34 ± 26 mmHg in the IH and normoxic groups, respectively, during pretest 1 (P = 0.40) and 27 ± 25 and 35 ± 16 mmHg in the IH and normoxic groups, respectively, during pretest 2 (P = 0.39). The vasocostriction index remained unchanged after chamber exposure (P = 0.15 and 0.16 for IH and normoxic groups, respectively), and it was not different between the groups (38 ± 18 and 25 ± 15 mmHg in IH and normoxic groups, respectively, P = 0.10), suggesting no carryover effects on sympathetic vasocostrictror responsiveness from 4 wk of intermittent hypobaric hypoxia in these athletes.

**DISCUSSION**

The major finding of the present study is that 4 wk of intermittent hypobaric hypoxia did not alter steady-state hemodynamics, cardiovascular variability, or cardiac-vagal baroreflex function in highly trained young individuals. These results do not support our hypothesis and suggest persuasively that this regimen of intermittent hypobaric hypoxia does not cause clinically significant abnormalities in BP control in young athletes. In contrast to animal studies, we found no evidence for sustained physiologically significant sympathetic excitation in this model.

**Effects of IH on autonomic function.** Previous studies have demonstrated that acute exposure to hypoxia leads to stimulation of the peripheral chemoreceptors, which in turn causes an increase in sympathetic tone (6, 7, 42) and a decrease in vagal tone.

### Table 3. Resting steady-state hemodynamics

<table>
<thead>
<tr>
<th>Variable</th>
<th>IH (n = 10)</th>
<th>Normoxia (n = 12)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre 1</td>
<td>Pre 2</td>
</tr>
<tr>
<td>SBP, mmHg</td>
<td>125±10</td>
<td>120±11</td>
</tr>
<tr>
<td>DBP, mmHg</td>
<td>72±6</td>
<td>68±5</td>
</tr>
<tr>
<td>HR, beats/min</td>
<td>71±11</td>
<td>68±11</td>
</tr>
<tr>
<td>Qc, l/min</td>
<td>7.6±2.06</td>
<td>7.23±1.69</td>
</tr>
<tr>
<td>SV, ml</td>
<td>111±23</td>
<td>108±28</td>
</tr>
<tr>
<td>TPR, dyn·s·cm⁻²</td>
<td>985±214</td>
<td>991±228</td>
</tr>
</tbody>
</table>

Values are means ± SD. SBP, systolic blood pressure; DBP, diastolic blood pressure; HR, heart rate; Qc, cardiac output; SV, stroke volume; TPR, total peripheral resistance. There were no significant differences in any variable within or between groups at any time.

### Table 4. Cardiovascular variability and baroreflex sensitivity during fixed breathing protocol before and after chamber exposure

<table>
<thead>
<tr>
<th>Variables</th>
<th>Hypoxia (n = 10)</th>
<th>Normoxia (n = 12)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre 1 (1)</td>
<td>Pre 2 (2)</td>
</tr>
<tr>
<td>SDRR, ms</td>
<td>68±27</td>
<td>66±33</td>
</tr>
<tr>
<td>LFRR, ms²</td>
<td>590±408</td>
<td>883±769</td>
</tr>
<tr>
<td>HFRR, ms²</td>
<td>2,960±3,699</td>
<td>2,825±3,213</td>
</tr>
<tr>
<td>NormLFRR</td>
<td>0.15±0.12</td>
<td>0.20±0.15</td>
</tr>
<tr>
<td>NormHFRR</td>
<td>0.43±0.24</td>
<td>0.44±0.21</td>
</tr>
<tr>
<td>LFBP, mmHg²</td>
<td>6.7±4.0</td>
<td>9.7±5.2</td>
</tr>
<tr>
<td>HFBP, mmHg²</td>
<td>17.4±14.3</td>
<td>13.3±13.2</td>
</tr>
<tr>
<td>GainLF, ms/mmHg</td>
<td>9.9±5.5</td>
<td>10.3±6.0</td>
</tr>
<tr>
<td>GainHF, ms/mmHg</td>
<td>10.2±5.2</td>
<td>11.1±6.5</td>
</tr>
<tr>
<td>CohLF</td>
<td>0.61±0.13</td>
<td>0.56±0.16</td>
</tr>
<tr>
<td>CohHF</td>
<td>0.75±0.10</td>
<td>0.73±0.15</td>
</tr>
<tr>
<td>PhaseLF</td>
<td>-0.21±0.45</td>
<td>-0.07±0.35</td>
</tr>
<tr>
<td>PhaseHF</td>
<td>-0.28±0.62</td>
<td>-0.15±0.39</td>
</tr>
</tbody>
</table>

Values are means ± SD. SDRR, standard deviation of R-R interval (RRI); LFRR and HFRR, power in low and high frequency of RRI; NormLFRR and NormHFRR, normalized power in low and high frequency of RRI; LFBP and HFBP, power in low and high frequency of SBP; GainLF and GainHF, low- and high-frequency transfer function gain between SBP and RRI; CohLF and CohHF, systolic pressure-to-RRI coherence at the low and high frequency; PhaseLF and PhaseHF, systolic pressure-to-RRI phase at low and high frequency. One- and 2-way repeated-measures ANOVA were used for comparisons within and between groups. There were no significant differences in any variable within or between groups at any time.
activity (6, 7, 37, 44), resulting in increases in HR, BP, and vasomotor sympathetic outflow to the muscular bed (42, 48), whereas TPR decreases because of the local vasodilation due to the reduced blood oxygen content (30). During continuous chronic hypoxia, a marked and long-lasting sympathoexcitation was found in healthy humans (25). Some investigators reported that sympathetic and parasympathetic activities remained constant (6, 37) or even returned progressively toward normoxic levels during chronic hypoxia (26, 44), presumably because of autonomic adaptation. However, the chronic effect of intermittent hypoxia in humans is difficult to study, because chronic cardiovascular and hemodynamic changes may take a long time to manifest (18). Thus results regarding the effects of chronic IH, especially the carryover effects on autonomic function in healthy individuals, are very few and controversial (34).

In the present study, we found that steady-state hemodynamics did not change after 4 wk of intermittent hypobaric hypoxia in young athletes. This finding is consistent with four previous reports (1, 5, 41, 55) but different from others. For example, in a study similar to the present investigation, but with shorter exposure to IH (90 min, 3 times/wk, 4,000–5,500 m for 3 wk), Rodríguez et al. (41) observed small decreases in BP at rest and during submaximal exercise. These results were in contrast to an increase in BP noted by Ekblom and Berglund (15) in subjects after administration of recombinant human erythropoietin.

Conversely, Bender et al. (3) showed that mean arterial pressure and TPR in hypoxia were increased after acclimatization during moderate-to-near-maximal cycle exercise in sea level-resident young men; moreover, Povea et al. (38) found that the low-frequency component and low-frequency-to-high-
frequency ratio of the HR variability during exercise in hypoxia increased, whereas the high-frequency component decreased, after 13 days of living at a high-training low training camp. However, in an investigation of the effect of two consecutive night exposures to hypobaric hypoxia on arterial pressure in healthy normotensive individuals, Arabi et al. (1) found that the carryover elevation of daytime DBP only persisted in the initial 60 min after exposure to hypoxia, whereas DBP returned to baseline by 90 min. Similarly, Xie et al. (55) showed that the increases in SBP and HR produced by intermittent asphyxia did not persist, although sympathetic outflow to skeletal muscle remained elevated in the postintervention recovery period, in healthy men. Tamisier et al. (49) found that the vasodilation during hypoxia persisted for ≥30 min after the stimulus and may contribute to the sustained sympathoexcitation after hypoxia.

Inasmuch as we did not measure muscle sympathetic nerve activity, we could not rule out the possibility that sympathetic outflow to skeletal muscle was elevated after IH in this study. Indeed, recently, Tamisier et al. (48) showed that muscle sympathetic nerve activity increased immediately after 2 h of sustained hypoxia exposure. However, neither DBP nor TPR changed, and, moreover, sympathetic vasoconstriction evaluated during the Valsalva maneuver was not altered, after chamber exposure in our subjects, indicating that the ultimate downstream effect of sympathoexcitation, i.e., arteriolar vasocnstriction, was not affected by 4 wk of intermittent hypoboric hypoxia. The unaltered cardiovascular variability and cardiac-vagal baroreflex function in all subjects after chamber exposure in the present study further support the notion that this regimen of intermittent hypoboric hypoxia has no carryover effects on BP control in highly trained athletes.

Intermittent hypoboric hypoxia is not sleep apnea. Although IH is commonly used as a model for sleep apnea in animals (16, 20, 36), there are concerns over its safety in humans. We found that 4 wk of IH did not change steady-state hemodynamics and baroreflex function in young athletes. These results indicate that this regimen of IH does not cause sustained physiological sympathoexcitation and hypertension and is safe in human research on athletic applications.

However, our model differs from the conditions encountered in patients with obstructive sleep apnea in the following aspects. 1) Apnea is associated not only with hypoxia, but also with hypercapnia and asphyxia, all of which lead to sympathoexcitation (19). In our study, subjects in the IH group did not truly experience “apnea,” rather, they experienced hypoxia. Thus our study is more comparable to previous studies at high altitude. 2) The duration of the hypoxia is different. Most sleep apnea is brief. Although IH is broadly defined as repeated episodes of hypoxia interspersed with episodes of normoxia, the actual protocols used experimentally vary greatly in cycle length, the number of hypoxic episodes per day, and the number of exposure days (34). It was found that, in humans, sympathetic activation was more likely induced by longer periods of hypoxia than by very short cycles (48). Thus, as reported by Bernardi (4), it is likely that sympathetic activation is related to the length and the number of subsequent repetitions of the hypoxic exposures at comparable hypoxic intensity. 3) Our population varied substantially from patients with sleep apnea. It has been demonstrated that athletes have increased vagal tone compared with their sedentary counterparts (17). 4) Our hypoxic stimulus was hypobaric, rather than isobaric, hypoxia. The hypoxia experienced in sleep apnea is most likely isobaric. Therefore, the specific role of IH in producing the major clinical consequences of obstructive sleep apnea has been difficult to sort out from clinical studies.

Baroreflex function assessment by the Valsalva maneuver. The Valsalva maneuver may be the most widely used test of baroreflex function in humans, and responses to this test have been studied extensively in healthy individuals and patients with cardiovascular diseases and autonomic dysfunction (14). The Valsalva maneuver holds great attraction, because it is safe and yields reproducible and quantitative results (2, 29).

Smith et al. (45) demonstrated that terminal arterial pressure elevations were proportional to the intensity of straining and to the preceding level of sympathetic firing. Because such pressure elevations are prevented almost completely by prior ganglionic blockade (56), proportionality between increases of sympathetic activity and subsequent increases of pressure probably represents a cause-effect relation. Despite the potential complexity of the Valsalva stimulus on aortic baroreceptors, thoracic aortic dimensions were found to decrease con-
sistently and in a highly reproducible fashion (45). Thus, ultimately, transmural aortic pressure declines in a consistent fashion during the Valsalva maneuver, which makes it such a reliable probe of autonomic function. These observations may have practical importance; in subjects in whom direct sympathetetic nerve recordings are not available, Smith et al. (45) suggested that “arterial pressure elevations after release of Valsalva straining provide acceptable estimates of preceding sympathetic nerve responses and also of the integrity of autonomic control mechanisms.”

In the present study, we found that cardiac-vagal baroreflex sensitivity or sympathetic vasoconstrictor responsiveness assessed by the Valsalva maneuver remained unchanged after 4 wk of intermittent hypobaric hypoxia in highly trained athletes. These findings further support the notion that this regimen of intermittent hypobaric hypoxia has no carryover effects on BP control.

Study limitations. There are at least five limitations in this study. 1) Autonomic control of BP was assessed not immediately but, rather, 3 days after the last chamber exposure in all subjects during quiet rest. It is possible that hemodynamics, cardiovascular variability, or baroreflex function could have been different between the groups immediately after chamber exposure or during exercise. It is also possible that there were opposite, but counterbalancing, effects on neural activity and vasoconstrictor responsiveness. However, the key clinical consequence from a safety perspective is whether such exposures lead to neural and vascular remodeling and sustained hyper-tension. Clearly, this was not the case in the present study. 2) Autonomic function tests were performed only before and after chamber exposure. We did not measure hemodynamics, cardiovascular variability, or cardiac-vagal baroreflex function during chamber exposure in our subjects. Therefore, we do not know whether autonomic function may change during hypobaric hypoxia in this model. 3) All subjects in our study were young elite athletes, which limits the generalizability of the findings. Further investigations with more subjects in different populations are needed to confirm this finding. 4) On the basis of the two pretest measurements, statistical power was calculated for each measurement. In general, power was 0.80–0.90 for most measurements within groups, but, for some more variable measurements, power was less. However, although individually some of these indexes may be relatively imprecise, together, they make a strong case that autonomic control of BP was not altered after 4 wk of this very intensive IH stimulus. 5) The interpretation of the results from the present study are specific to this severe, 3-h hypobaric hypoxia paradigm and perhaps cannot be extrapolated to other models of IH used by athletes.

In summary, there were no significant differences in steady-state hemodynamics, cardiovascular variability, or baroreflex function in the IH or the normoxic group at any time. Our results suggest that exposure to intermittent hypobaric hypoxia for 4 wk has no carryover effects on autonomic control of BP in young athletes. This indicates that the use of intermittent hypobaric hypoxia as a modality to improve exercise performance is safe in this population. In contrast to animal studies, we found no evidence for sustained physiologically significant sympathoexcitation in this model.

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